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LECTURES ON

CLINICAL MEDICINE,

DELIVERED AT THE HÔTEL-DIEU, PARIS.

BY

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TRANSLATED AND EDITED

WITH NOTES AND APPENDICES

BY

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ON VENESECTION IN CEREBRAL HÆMORRHAGE AND APOPLEXY.

Apoplexy is not to be confounded with Hæmorrhage.—Cerebral hæmorrhage rarely sets in with apoplectiform phenomena, properly so called.—Apoplexy may be the expression of various grave lesions of the Encephalon.—Value of facial hemiplegia in Hæmorrhage.— Inutility of venesection, of bloodletting in general, of purgatives and emetics in hæmorrhages and apoplexy.—Differential diagnosis between softening and hæmorrhage.—Value of certain signs with regard to prognosis.

Gentlemen,—The patient lying in bed No. 7, St. Agnes ward, affords me the opportunity of raising a question of the highest clinical importance, namely, the contra-indication of bloodletting in cerebral hæmorrhage, and more generally in apoplexy. I will at the same time draw your attention to the semiotic value of facial paralysis when it occurs in connection with a lesion of the opposite hemisphere of the brain, and not as a consequence of disease exclusively limited to one of the facial nerves. Lastly, I will say a few words respecting the differential diagnosis between softening of, and hæmorrhage into, the brain.

The patient, whom I mentioned just now, was admitted into the hospital for a chronic pulmonary catarrh, which had not caused any notable disturbance of his general health. He was under treatment for that complaint, when he was suddenly seized with symptoms which caused me some anxiety, although he did not himself complain of them.

Without any premonitory headache or giddiness, he found a few days ago that his tongue was embarrassed, and that his speech was thick. His intellect was not in the least affected, his sight was perfect, his activity and his muscular strength were not diminished in the slightest degree; for his legs, at least, carried him, and moved as usual, and his gait was

not vacillating. Having had occasion to write, however, he noticed, as soon as he took up his pen, that he had some difficulty in using it, and that his letters were not formed so well as usual. These symptoms excited so little anxiety in him that he complained to nobody, and that at my visit, on the following morning, he did not think of mentioning them. On coming near him, I was struck, however, with the alteration in his features; for there was evident deviation of his mouth. I questioned him, and then heard of the above-mentioned symptoms, which he had noticed on the previous day. You heard him state and repeat that no intellectual disturbance, no affection of the senses, preceded or accompanied this thickness of speech, of which he was perfectly conscious, and the awkwardness with which he used the fingers of his right hand.

You could study the character of the deformity of his face,—how, on the left side, the labial commissure was markedly pulled upwards and outwards, whilst it was lower on the right, the corresponding cheek being at the same time flattened and almost motionless. At first, you might have thought that there was also deviation of the tongue, for when the patient was asked to protrude it, the organ seemed to incline to the right of the middle line; but this deviation was only apparent, and was due to a change in the normal relations of the tongue to the aperture of the mouth from the pulling outwards, and to the left of the labial commissure. The paralysis did not involve the face alone, for besides the awkwardness in writing noticed on the preceding day, there was weakness of the whole upper extremity on the right side, and he added, also, that he had had, that very morning, tingling sensations in the tips of the fingers of his right hand, which had lasted a minute or two. The sensibility of the skin was perfect and normal.

Now, what is the matter with this man? I have no doubt that he is suffering from the effects of a small hæmorrhage into the left hemisphere of the brain. Yet I acknowledge that, at first sight, the diagnosis offered some difficulty, for the case might have been thought one of facial paralysis only. It was the face that was chiefly affected, and the power of motion of the lower limb was perfect according to the patient, who averred that he noticed no difference between his right and left legs, and that on both sides he possessed as much strength as before. There was, indeed, undoubted paralysis of the right arm, but very slightly marked, involving limited movements only, and even then, it was on having occasion to write that the patient noticed the deficiency in the suppleness of his fingers. Not so with the paralysis of the face; this was evident to all bystanders, more so than to the patient himself, who was so unconscious of it that he did not complain of it. In this paralysis of the face,

however, we already possessed an element of great value for making the diagnosis of *Cerebral Hæmorrhage*, which I wrote on the card, because this paralysis of the facial muscles was not so complete as it usually is when it depends exclusively on disease of the seventh cranial nerve.

In facial paralysis, caused by a lesion of one hemisphere of the brain, whether attended or not by paralysis of the limbs on the same side, the patient cannot perform with ease certain movements on the affected side, such as the act of blowing or getting back into the cavity of the mouth food which has lodged between the cheek and teeth, but he is not completely incapable of performing such movements, and the difficulty he experiences is never so great as that felt by individuals suffering from pure facial paralysis. In the former case, also, the orbicularis palpebrarum is never paralyzed to the same extent as in the latter; hence, if a hemiplegic patient be asked to shut his eye, he does it completely enough to hide the globe of the eye, whilst the eyeball remains uncovered in cases of paralysis of the seventh pair.

I do not attempt to find a reason for this difference; I merely note it as a fact taught me, long ago, by experience, and the importance of which, in making a differential diagnosis, you will immediately recognize. Thus, in this patient, the incompleteness of his facial palsy, in the absence even of other characteristic phenomena of a more extensive hemiplegia, sufficed to lead me to believe that the paralysis was due to some lesion of the left hemisphere of the brain, and not to disease of the portio dura.

But this, gentlemen, is not the most essential point to which I wished to direct your attention. The patient had, I say, cerebral hæmorrhage to a small amount. Observe that I do not use the word apoplexy, and purposely so, because there is a great difference between cerebral hæmorrhage and apoplexy, although some confound them still, in spite of the majority of our classical authors who try to do away with this deplorable confusion. Now, what is meant by *apoplexy?* According to its etymology, it means an affection in which, as the ancients described it, an individual falls, and is struck down suddenly, like an ox felled by the butcher. "*Apoplexia dicitur adesse quandò repentè actio quinque sensuum externorum, tùm internorum, omnesque motus voluntarii abolentur, superstite pulsu plerùmque forti, et respiratione difficili, magna, stertente, unà cum imagine profundi perpetuique somni.*" And if to this short sketch of apoplectiform phenomena, given by Boerhaave, you add the definition of Paulus Egineta, that this abolition of consciousness and of the sensibility of the whole body is caused by an affection of the *sensorium commune* (*communi nervorum principio affecto*), you will know what is meant by apoplexy

You understand now why this term and that of hæmorrhage should not be considered as synonymous. On the one hand, *apoplexy* is a generic term which must be specified, because apoplectiform phenomena are often connected with pathological conditions very different from hæmorrhage. Thus, they may be the result of *cerebral softening*, of a rapid and more or less considerable accumulation of serosity in the ventricles and in the cerebral meninges, as in what is called *serous apoplexy*; or they may be due to *congestion* carried to the highest point, without actual extravasation of blood, as in what is termed *ictus sanguinis* (but in the next lecture I will show you how rare such cases are); or again, apoplexy may be produced, as the ancients had already noted it, by what we now term *embolism*. Lastly, it sometimes occurs independently of all appreciable lesion, on dissection, in the so-called *nervous apoplexy*. On the other hand, cerebral hæmorrhage is not necessarily accompanied by symptoms of apoplexy; these show themselves only when the hæmorrhage is pretty considerable. Small hæmorrhagic clots can be formed, not only without the patient presenting the series of phenomena constituting apoplexy, but without his having any impairment of intellect, any affection of the senses; in fact, without any symptom indicating that the brain has been deeply modified in its functions. The only symptoms which then characterize the case are those of paralysis, more or less complete, and more or less limited in extent on the opposite side of the body.

During the period that I have been in the habit of delivering clinical lectures at the Hôtel-Dieu of Paris, I have only seen one female and two male patients in whom cerebral hæmorrhage *seems* to have set in at once with apoplectiform phenomena. You doubtless remember that rag-collector who was found in the streets, and brought to the hospital in the most profound stupor, and laid in bed No. 5, St. Agnes ward. He died on the second day after his admission, and when his brain was placed on the amphitheatre table, I announced to you that we should find an effusion into the ventricles. It turned out that the blood had been first poured out in one of the corpora striata, from there had passed into the lateral ventricle of the same side, and after filling it, had broken down the septum lucidum, and got into the other lateral ventricle.

During the summer of 1861 you saw in the St. Bernard ward also, a woman, aged 63, who had had, the preceding year, a so-called paralytic stroke. She had faltered in her speech all of a sudden, and had been seized with weakness of one half of the body. There was, on that occasion, no loss of consciousness, no giddiness even. This time, she was found in her bed in a state of profound coma. She died without having been roused, and, as in the last case, there was found, in addition to the

remains of the small hæmorrhage of the previous year, an enormous clot, beginning in one optic thalamus, and distending both lateral ventricles.

Again, you may still recollect that young man lying in No. 15 bed, St. Agnes ward, who, whilst presenting all the symptoms of encephalitis, was suddenly seized with epileptiform convulsions, and died a few minutes afterwards in a state of carus. In this case there was hæmorrhage into the pons Varolii, which had made its way into the fourth ventricle, and ruptured the valve of Vieussens. I repeat, gentlemen, apoplexy proper is very rare in cerebral hæmorrhage. You have seen at No. 34, in the St. Bernard ward, a very intelligent woman, 49 years old, who relates with perfect clearness her sad history. She was enjoying excellent health, when she noticed, one morning about eight o'clock, an impediment in her speech, and some numbness of her leg and arm. Feeling anxious at this, she walks down stairs from the third floor, and goes to a neighbouring chemist's shop. She there takes a few drops of ether, and returns home with less facility, feeling the numbness rapidly increasing. On reaching the bottom of her stairs, she is unable to proceed further, tries to prevent herself from falling by resting against the wall, but drops down, nevertheless, without losing consciousness, or even feeling in the least giddy. Her neighbours came to her help, and brought her to the Hôtel-Dieu. She was paralyzed on the right side.

You have not forgotten, either, the woman lying at No. 10, St. Bernard ward. She had just prepared and served the family dinner, at four o'clock in the afternoon. She was eating with a very good appetite in company of her husband and children, without any headache or other premonitory symptom that attracted her attention. All of a sudden she finds that she cannot cut her bread; she says so to her husband, but with a thick voice. She tries to get up, and falls down with her chair, without losing consciousness or having felt giddy. As she is raised up she is found to be hemiplegic, and on her admission into the hospital she relates herself the above details, with perfect clearness, and even with a certain degree of cheerfulness.

I insist on these two cases, because of the fall of both patients, the one on trying to get up from her chair, the other on reaching the foot of the stairs. This fall, I beg you to observe, differs essentially from that of a person struck down by apoplexy, but is analogous to the fall of a soldier whose leg is broken by a ball; inability to move the leg, and its extreme weakness, being the essential cause in both cases. The intellect is not affected, as it is in the apoplectic attack of epilepsy or of eclampsia. In the latter case, the individual drops like an ox knocked down by the butcher, and the phenomena which ensue are really those

of apoplexy, such as described by our predecessors, phenomena which are observed in cases of cerebral hæmorrhage only when there is effusion of blood into the ventricles, or the pons Varolii, or to an enormous amount in the centrum ovale of Vieussens, or again, into the arachnoid sac.

I made use, just now, gentlemen, of a very restricted form of expression, when speaking of the rag-collector who had been picked up in the streets in a state of *apoplexy*. I told you that the cerebral hæmorrhage *seemed* to have set in with apoplectic symptoms, as if I had some reason for doubting the accuracy of the fact. I doubt it, indeed, for if it be undeniable that the man was picked up and brought to the Hôtel-Dieu in a state of apoplexy, and that the old woman I just mentioned was found one morning in her bed in a state of coma, who can positively affirm that these symptoms of apoplexy set in all of a sudden?

In the spring of 1863 I was asked by my friend Dr. Marchal (de Calvi) to see a man, aged 63, who had had a fit that same morning. Whilst at breakfast, he had suddenly found some difficulty in holding his fork, and had felt slightly giddy. On attempting to speak, he noticed himself that his speech was thick, and his children made the same observation. He staggered as he rose, felt weaker on one side than on the other, but, with the help of his son, managed to walk as far as his bedroom. He was then undressed and put to bed, he, all the time, understanding perfectly all that was being done, without any impairment of intellect, nor were his movements abolished. The hemiplegia made rapid progress, however, and became complete within half an hour. The intellect got gradually more and more clouded, and when Dr. Marchal arrived, half an hour or three-quarters of an hour after the setting in of the first symptoms, the patient was already in an *apoplectic* condition. Things went on from bad to worse, and when I came myself at five o'clock in the evening, the apoplectic stupor was at its height. In spite of the most energetic treatment, the patient died in the night.

About the same date, I was fetched to see a patient of Dr. Revilloux, a man about 62 years old, who noticed, whilst at dinner, that one of his hands felt heavy; he was not giddy, and only faltered in his speech. He tried to rise from his chair, but one of his legs being paralyzed, he fell down, without losing consciousness however. His children lifted him up, and with their assistance he walked as far as the next room, and there sat on a chair. I arrived three-quarters of an hour after the manifestation of the first symptoms. The patient retained, or seemed at least to retain, all his intellect. He answered me to the point, although his tongue was very much affected; and his left arm and leg were almost completely paralyzed of motion. Pro-

found coma set in a few hours later, and death occurred the following morning.

Very recently again, I admitted into the St. Bernard ward a woman, aged 56, who had formerly been subject to the periodic headaches of gout, and who, eight months previously, had been seized one morning with the first symptoms of cerebral hæmorrhage. She had gone out marketing, in as good health as ever; on returning home she noticed that she dragged her right leg, and that her right arm felt heavy. She even changed to her left hand a folded newspaper which she was carrying home, for fear of dropping it into the mud. She walked upstairs to her room, undressed, and got into bed. To questions of her husband, she replied in a faltering voice. The symptoms growing hourly worse, she became completely hemiplegic, and partially unconscious towards evening. About twelve hours after the setting in of the illness, and for three days, she remained in a state of profound stupor. This case is interesting from other points of view also, and I shall return to it later; for, contrary to what usually obtains, the patient regained the power of moving her arm much more quickly and more completely than her leg, and I shall tell you what is the value of this sign.

But to return to my proposition. In the case of the rag-collector and that of the old woman I spoke of before, who knows how the attack set in? who knows whether for half an hour, an hour, and even more, the symptoms had not run the same slow and progressive course as in the three cases I have just related to you? Nay, I add that this is infinitely probable, if not absolutely certain. The reason why I speak so positively is, because for more than fifteen years my attention has been directed to this point in the history of cerebral hæmorrhage, and I never had the chance, *never once*, of seeing a patient struck down suddenly by *apoplexy*, in the classical and etymological sense of the word. I have not seen a single case in my hospital or my private practice, or in the practice of my professional brethren who have done me the honour of asking me to meet them in consultation. I have, indeed, seen a great number of individuals suffering from cerebral hæmorrhage, in the most profound apoplectic stupor; but in every case, *without exception*, when the attack had occurred in presence of witnesses, it had come on gradually, and had in general been slight at the outset, coma supervening ten minutes, half an hour, an hour, or several hours afterwards; but in no single instance, I repeat, have I seen a man with cerebral hæmorrhage struck down as by a blow, and dropping instantly in a state of unconsciousness.

Under certain circumstances only is this the case, and I hasten to make the statement, lest my views should be deemed exaggerated or singular. The patient in No. 15 bed, St. Agnes

ward, who died of hæmorrhage into the pons Varolii and tearing of the valve of Vieussens, became suddenly comatose, and remained so until his death, which occurred shortly after. But what did his night attendant tell us? The patient, you remember, had acute encephalitis, that would have carried him off a few days later had not this unforeseen attack occurred. All of a sudden he is seized with epileptiform convulsions, and he dies a few minutes afterwards, without having been roused from the most profound apoplectic stupor. Note well, gentlemen, that to the ordinary phenomena of hæmorrhage there was superadded, in this case, an attack of convulsions, which alone, and apart from all complications, suffice for producing apoplectic stupor. I admit, then, that whenever cerebral hæmorrhage begins with an epileptiform attack, apoplectic stupor will set in suddenly, as it does after every attack of epilepsy. I will add further, with regard to this case, that the hæmorrhage was seated in the pons Varolii, that is, in a point where all the nerve-fibres converge. When hæmorrhage occurs in a part so essential to life, I understand the suddenness of apoplectiform phenomena. But again, I repeat, apoplectic stupor is a very exceptional symptom of invasion in cases of cerebral hæmorrhage, unless there be lesion of a central part, or an attack of convulsions.

I make no exception even in favour of blood-effusion into the lateral ventricles. Before this happens the blood has accumulated in a portion of the brain, near the surface of the ventricles, and has already given rise to symptoms which may have been mistaken, but which indicate, to the experienced practitioner, the existence of hæmorrhage, or of a morbid process which has caused capillary hæmorrhage. Suppose, for example, that such a morbid process takes place in a corpus striatum, and that in consequence of it a number of small clots have formed, varying from the size of a small pin's head to that of a small lentil, so far there will only be a sensation of weight in the head, and of numbness in the side opposite to the lesion; but if, all of a sudden, on the blood finding its way into a ventricle, the person falls down, struck with apoplexy, the symptoms noticed before the occurrence will be considered as premonitory only, whilst they were in reality symptoms of a simple or multiple hæmorrhage, dating a few days back. In such a case the hæmorrhage is supposed to occur only when the patient becomes apoplectic; whereas the blood is effused into the cerebral substance at the time the first symptoms manifested themselves, the subsequent formidable accidents being caused by the sudden irruption of the blood into the ventricles.

You saw what happened in the case of the patient who forms the subject of this lecture. He had no warning when the hæmorrhage began, and even after its occurrence there was nothing serious enough to excite his anxiety. He had only some impedi-

ment in his speech, some difficulty in writing, which alone attracted his attention, and a deviation of the mouth, of which he was not conscious before I observed it. If the suddenness with which the symptoms showed themselves, and their truly hemiplegic character, although the hemiplegia was limited to the face and the right arm, led us to infer that hæmorrhage had taken place in the left cerebral hemisphere, the slight degree of impairment of motor power led us also to believe that the clot was very small, probably of the size of a lentil or a cherrystone. Now, such hæmorrhages are not, by themselves, followed by fatal results, although they sometimes, it is true, indicate an unpleasant organic predisposition to the recurrence of similar accidents. By this organic predisposition I do not mean softening of the cerebral substance, which, according to Rochoux, necessarily precedes hæmorrhage, and which he accordingly considers as paving the way to it, and terms "hæmorrhagipare," nor those changes in the cerebral vessels to which Abercrombie attributed the greatest share in the production of hæmorrhage. Agreeing in this with the majority of medical men, I believe that the softening of the brain which accompanies hæmorrhage is an effect, and not a cause. Its importance, however, is not the less great, for much more frequently than the hæmorrhage itself, the sequential acute softening, the encephalitis, is the cause of grave cerebral accidents, and ultimately of the patient's death. As to the changes in the coats of the cerebral arteries, such as yellow laminæ of cartilaginous consistency, mostly impregnated with calcareous salts, they cannot be an essential condition for the production of cerebral hæmorrhages, since they are not met with in the greater number of cases, although present in some, as I have shown you instances.

To return to our patient in the St. Agnes ward, the symptoms in his case were so mild, that we were authorized to suppose the cerebral lesion to be unimportant, and to hope that the case would turn out favourably. Indeed, the man leaves the hospital to-day, feeling well enough to resume his usual occupation.

Perhaps you have been surprised to see me do nothing in this case; and have you asked yourselves why, when so many others would have hastened to employ active treatment and had recourse to bleeding, either local or general, or both, purgatives and revulsives, I simply did nothing? Those who have seen my practice for some length of time have been less surprised, because they know that I never use violent remedies, that I not only abstain from all energetic treatment when the symptoms of cerebral hæmorrhage are as slight as they were in this case, but that I even refrain from doing so in very grave cases, in fact, in all cases of apoplexy.

My reasons are these:—If I do not have recourse to blood-

letting, purgatives, or revulsives in cerebral hæmorrhage, whether considerable or not, it is because experience has taught me that the patients do better without them. For when I reflect on what happens then, I do not see how those methods of treatment can be of any use, since the hæmorrhage is an accomplished fact when we are called upon to note its symptoms. What influence, I ask, can be exerted on a foreign body in the shape of extravasated blood, by letting out blood from a vein of the arm, or of the foot, or from the jugular, or by dividing an artery, by cupping, or leeches? Of what use are purgatives or revulsives? It is said that bloodletting, and that purgatives, a kind of serous bleeding, empty the vessels, and thus facilitate absorption of the extravasated blood; that they antagonize the cerebral congestion, which, according to the practitioners who recommend them, precedes, accompanies, or follows, at the least, the extravasation of blood, and by thus preventing an exaggerated flow of liquid, they diminish the risks of the effusion becoming more considerable or occurring a second time.

With regard to the first point, we may well doubt whether any difference obtains between cerebral hæmorrhages and other hæmorrhages, and, to take a very simple example, whether any difference exists between what takes place in cases of extravasation of blood into the cerebral substance and extravasation under the skin. In the latter case, has general or local bloodletting ever been seen to facilitate the absorption of the effused blood? Do not most surgeons reject leeches, on the contrary, as being injurious, instead of useful? An individual receives a blow, or falls on his head for example, and the violent contusion produces a more or less considerable effusion of blood into the subcutaneous cellular tissue. Any medical man who may be sent for, will never think of prescribing anything more than cold lotions on the affected part, or using slight compression; and he does so, because he knows full well that further interference would, to say the least, be superfluous. Now, can we act more powerfully on *ecchymoses* of the brain than on those of the surface of the body? Reasoning therefore, agreeing with experience, pronounces useless the treatment against which I raise my voice.

As to the second point, namely, that bloodletting is imperatively required with a view of arresting the molimen hæmorrhagicum which caused the first symptoms, and might cause them a second time, it is indeed very doubtful. The part played by congestion seems to me to have been very much exaggerated, and although a great many practitioners believe general or local bloodletting to be so clearly indicated that there need be no hesitation in having recourse to it, I do not think that the necessity, nay more, the usefulness of the measure, has been clearly proved.

Do we know well the organic conditions under the influence of which cerebral hæmorrhage is produced? That congestion sometimes accompanies it, is a fact generally accepted; but is not this an effect rather than the cause of the extravasation of blood? What influence then can bloodletting exert on this sequential hyperæmia, when it has none on the foreign body formed by the effused blood, which is the starting-point of the determination of blood? Furthermore, far from being useful, bloodletting has seemed hurtful to me, and I believe that it favours instead of preventing congestion. In the next lecture I purpose studying apoplectiform cerebral congestion, and I shall then tell you how I understand what occurs in apoplexy, and I shall speak of what I term *cerebral surprise*. I hope to be able to show you that apoplectic phenomena are in some measure more allied to syncope than to congestion, and that bleeding is therefore contra-indicated, not demanded. This is what experience has taught me, and has taught others who follow in their practice the same rules as I do.

What treatment then do I adopt in cases of cerebral hæmorrhage, and more generally in apoplexy? Instead of bleeding my patients, of putting them on low diet, and keeping them in bed, I do not draw blood from them, I recommend to them to get up if possible, at least to remain in the sitting posture, and I feed them. I am convinced that I thus obtain much more favourable results than when I interfered more actively, and that patients so treated do a great deal better than those whom I bled in former days, kept on low diet, and confined to their beds.[1]

I reject bloodletting from the treatment of cerebral hæmorrhage, although I think that very plausible reasons are urged by those who act differently from me. I did myself for a long time

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[[1] On this important question, consult Dr. Todd's "Clinical Lectures on Nervous Diseases." "There is a practice unfortunately too common," says this eminent physician, "but which, I think, is every day becoming less common—namely, that of following an attack of apoplexy by depletive measures very much as a matter of course. The case, indeed, which I have just detailed to you, is one of many which proclaim loudly that a depletory system ought not to be pursued indiscriminately, or '*even generally*' in apoplectic cases. E. Copeman ('Collection of Cases of Apoplexy,' London, 1845) shows that considerably more than half of those treated by bleeding died" (p. 117, *op. cit.*).

Again, at pages 119 and 120, Dr. Todd says: "If, upon full inquiry into all the particulars of the case, you find that your patient is of full plethoric habit, with too much blood in his body, and with a sufficiently strong heart, you may bleed him with every chance of success; but if he has been of intemperate habits, is labouring under organic disease of the heart and arteries, is of gouty or rheumatic constitution, then, whatever popular or medical custom may say, my advice to you is, hesitate much before you deplete by bleeding . . . ."

"Bear in mind that in a large number of cases—probably the majority—there is in reality no cerebral congestion, and that the hæmorrhage is not of a kind likely to be stopped by taking away blood."—Ed.]

what most practitioners still continue to do now, and I used to think my plan very rational. I may add that, in spite of ourselves, we feel the influence of fashion, however sad the confession may be. I began practising medicine at a time when the doctrines of Broussais were in all their glory; and although I had been a pupil of Bretonneau, who had dealt the heaviest blows to the doctrines of the illustrious physician of the Val de Grâce, I felt not the less the powerful influence of those doctrines, and I was induced to prescribe leeches in cases where I never think of doing so now, merely because everybody did it, and because no amount of self-confidence can make one believe he is right when he is in opposition to everybody else. I bled, therefore, in cerebral hæmorrhage, because bleeding was used before and around me. Now that I have reached an advanced age and that I occupy a position which allows me freely to follow my inspirations, I still understand how a young practitioner has neither courage nor self-confidence enough to reject a mode of treatment which has been in some measure sanctioned by the experience of several generations of medical men.

But there is another circumstance which renders non-interference still more difficult,—I mean, the febrile action, which rarely fails in hæmorrhages of a certain amount. This febrile action, on which classical authors lay too little stress, usually commences from twenty to twenty-four hours after the outset of the attack, and reaches its maximum on the second or third day. The pulse becomes hard and frequent, the skin hot and often bathed in perspiration, the face flushed, respiration laboured. I confess that I have been induced to bleed under those circumstances when I had refused to do so in the beginning; but I must confess also that the bleeding has never seemed of any use to me; that it has often been manifestly injurious, and that if I had the courage to resist the seemingly pressing indication, the fever ceased, and the patient regained his strength with a much greater rapidity than if bloodletting had been had recourse to. In such cases, I still better understand how difficult it is for a young practitioner not to yield to the apparent urgency and to the entreaties of the friends who ask for bleeding, as well as the advice of brother practitioners who regard it as necessary. And as in a certain number of cases, this fever, lighted up on the second and third day, and the cause of which I cannot well explain, only ushers in formidable brain symptoms which become rapidly fatal, I understand that antiphlogistics may be thought of, although they prove useless, alas! when the disease runs the course I just mentioned. To save your responsibility in such cases, avoiding at the same time what your conscience forbids, open a vein, but in such a way as only to draw an insignificant quantity of blood and explain to the friends that it would be

dangerous to go further. In many cases there will be real danger in doing so, for some persons have been seized with fearful symptoms even after a moderate bleeding. A short time ago, a gentleman, a former pupil of mine, was sent for to a magistrate who had just been struck down with cerebral hæmorrhage. There were well-marked hemiplegia, distortion of the face, and impairment of speech; the intellect was perfect. Although he was of opinion that bleeding was not required, he was compelled to yield to the consulting physician, who had over him the superiority of age and of a high scientific position. The patient was therefore bled; but he had scarcely lost 100 grammes of blood (about three ounces) when he fell into a state of complete resolution, from which he never rallied, until his death, which occurred a few days afterwards. But a moment before the bleeding he was in the full enjoyment of all his faculties, and conversed freely and ably with his friends around.

I am not the only one, gentlemen, who regards bleeding, and the other means usually recommended in cerebral hæmorrhage and apoplexy, as useless and inconvenient. Very recently one of my colleagues, Professor Monneret, declared that he had for a long time given up the active treatment which, like myself, he formerly had recourse to. Far from lowering his patients, he feeds them, and gives them wine. Since I have conformed to the rule of keeping up the strength of my patients by giving them food in moderation, I find that the bad symptoms under which they labour disappear more rapidly than when I interfered actively, and the case you have lately had occasion to observe is another proof in favour of my assertion.

In the case of the patient in the St. Agnes ward, there supervened, in the course of his illness, certain phenomena to which I wish to draw your attention. You often heard him complain of attacks of giddiness, which were more or less prolonged; and, doubtless, many among you looked upon them as symptoms of a determination of blood to the head, and concluded that if I had bled the patient I would have avoided those threatenings of returning hæmorrhage. On carefully questioning the man, however, I found that the giddiness came on more frequently when he had been fasting, and ceased immediately on his taking food. It was not cerebral congestion, therefore, at least, congestion as it is generally understood, which caused the symptoms I alluded to. These were due to a deficiency in the normal constituents of the blood, unfitting it for stimulating the brain, and not to an excess of blood in the vessels. Bleeding would have aggravated this vertiginous disturbance, whereas nourishment speedily got rid of it.

Since we are on the subject of cerebral hæmorrhage, allow

me, gentlemen, to take the opportunity of speaking to you of softening of the brain, and of answering, to the best of my ability, the questions you often ask me in the course of my visits round the wards, embarrassing questions though they be, because you expect me to solve one of the most difficult problems in pathology, namely, the differential diagnosis between cerebral hæmorrhage and softening. Lying at No. 18, in our male ward, is a patient whose history is interesting from this very point of view. He was admitted into the hospital a few days ago, suffering from complete hemiplegia of the right side. His history is very short, and is as follows:—He was seized in the midst of the most perfect health, with the exception that for the last eight or ten days he had suffered from occasional giddiness and headache, and had felt confused at times. He had also noticed a sensation of numbness in his right hand and foot. He was not, however, prevented from walking or moving about, and attending to his usual occupation, when suddenly, a few days ago, he was struck with palsy of the right side. He then came to the Hôtel-Dieu, where I found complete paralysis of motion, with relaxation of the right arm and leg, involving the corresponding half of the face, besides nearly absolute anæsthesia of the integuments of the affected parts, marked dulness of aspect, and slowness of speech. The patient was free from fever.

I thought that cerebral hæmorrhage had occurred in this case, although I felt some hesitation at first, because of the complete loss of the power of motion, and the thorough resolution of the limbs on the right side, that were scarcely proportionate to the small degree of intellectual disturbance. Indeed, it does not usually happen, in my opinion at least, that in cerebral hæmorrhage there should be such complete paralysis of motion as there was in this instance, without there being loss of consciousness also. Complete loss of motor power, without accompanying coma at the time of seizure, belongs, I believe, more especially to softening. On many occasions, and for many years, I have specially called your attention to these elements of a differential diagnosis between hæmorrhage and softening, diagnostic characters laid down by Récamier, and for which I claim no credit to myself. According to my illustrious teacher, the value of actual symptoms is infinitely greater than that of the phenomena which in some cases precede the attack, although he does not deny that they are of some value. Récamier, indeed, affirmed, and in many cases I have been enabled to verify the truth of his proposition, that whenever hemiplegia, complete and absolute, occurs *suddenly* (and I insist on this point—the suddenness of attack), without loss of consciousness, softening of the brain may

be diagnosed.[1] Whenever, on the contrary, the complete loss of motor power is attended by loss of consciousness, whenever, especially, the individual has become suddenly comatose, hæmorrhage may be diagnosed, and hæmorrhage to a considerable amount. But when the intellect is affected to some extent, but not entirely—when there is obtuseness, but not complete loss of sensibility—whilst there is absolute loss of motor power, as in the case of our patient in St. Agnes ward, we must always, according to Récamier, diagnose hæmorrhage in connection with softening, or what has been termed capillary hæmorrhage. This latter form usually takes place in a softened portion of the brain, and is characterized, on dissection, by the presence either of a large number of small clots, perfectly isolated from one another, or coalesced so as to form larger hæmorrhagic centres. In those cases, but in them alone, was the eminent physician of the Hôtel-Dieu disposed to admit the antecedent softening which is by Rochoux regarded as the organic condition, the morbid process which must of necessity, and in all instances, precede cerebral hæmorrhage. I am too much a pupil of Récamier, I confess, not to adopt his conclusions, which my personal experience seems to me, besides, to have corroborated, in the case I have just alluded to. I am therefore inclined to diagnose hæmorrhage connected with softening of the brain. The grounds on which I rest my belief are, that the patient never suffered from the grave disturbance of the intellect, the loss of consciousness, the coma, or the somnolence at least, which usually accompany hæmorrhage of great magnitude; that he only felt a little confused, bewildered, and stupid, which symptoms coincided with a diminution in the cutaneous sensibility of the side, which was completely paralyzed of motion.

When speaking to you of the female patient lying in bed No. 11, St. Bernard ward, I said that I wished to call your attention to an unusual symptom which she presents, a symptom to which, I believe, sufficient importance is not given, as influencing prognosis. She told you, and we verified her statement, that she could move her arm better than her leg, and she added that for a few months after her seizure she had walked much better than she does now. You know that the reverse usually obtains, and that in the great majority of instances the lower limb regains the power of motion much quicker than the arm. Why it

[1 "I apprehend that it may be pretty confidently affirmed, when there is simply paralysis without coma, occurring *suddenly or with great rapidity*, that it is always dependent on softening without or with clot; that the softening is of the colourless kind; and that when a clot exists, it is so small as not to exercise pressure on neighbouring healthy parts of the brain." (Todd's "Clinical Lectures on Nervous Diseases," p. 207.)—Ed.]

is so I do not know, and I am not aware that anybody has ever given a satisfactory explanation of it. This is remarkable, however, that when the arm regains power quicker and better than the leg, the patient is worse off than when the reverse obtains.

Three years ago, I was sent for to see a general officer, a near relative of mine. He had been seized that morning, a little before breakfast, with paralysis of the right side. For three or four days after this his symptoms looked unfavourable, but fever soon ceased, and a fortnight after the attack he could write, shave himself, and walk pretty well. The extreme precision of the movements required for writing and shaving showed clearly enough that his arm was considerably better than his leg, for he walked very lame. After the lapse of a few months his leg became stiff and painful, and he walked with more difficulty. A stick was no longer enough for him, and he required the help of a friend's arm; later, even with this help, he was unable to walk. At that time the arm itself began to lose power, and the intellect failed in proportion. Subsequently, the poor man could not leave his arm-chair, and suffered excruciating pain in the paralyzed side, especially in the leg. He at last died in a state of perfect imbecility.

The same fate awaits the woman in the St. Bernard ward. She, too, uses her arm much better than her leg; but already, for the last two or three months, her leg, at night especially, has become acutely painful. For two months after her seizure she walked pretty well, whereas, now, she cannot take a single step unless strongly supported, and before another two or three months shall have elapsed, she will probably not be able to leave her arm-chair, and she will die within the year consumed by pain, and a thorough imbecile.

Now, gentlemen, if you ask me why our prognosis should be unfavourable when the arm regains power more completely and more rapidly than the leg, I must confess my ignorance, and content myself with noting a fact which has often enough occurred in my practice to have attracted my attention. I cannot say whether a morbid process goes on round the clot, causing chronic softening or irregular cicatrices; but whatever the cause may be, the fact remains, and seems to me to possess some value.[1]

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[1] [Dr. Todd (*op. cit.* p. 206) mentions the unusual fact of the arm regaining power before the leg, but does not seem to look upon it as an unfavourable symptom, since he speaks of it in connection with cases of recovery. That such cases are of very uncommon occurrence will appear from the fact that for the last eighteen months I have in vain looked for an instance of the kind among the numerous patients who attend the National Hospital for Paralysis and Epilepsy. Dr. Ramskill, the senior physician, however, informs me that a case came under his observation in private practice, presenting exactly the same characteristics as those described by Prof. Trousseau. The patient,

I will not leave this subject without calling your attention to another sign, which, like the preceding, is of great prognostic value. You doubtless remember two women, the one, still young, lying in No. 34 bed, St. Bernard ward, the other, aged 64, lying in bed 28. They were both paralyzed on the left side after an attack of cerebral hæmorrhage. There had been no impairment of intellect, and they both could walk before a month had elapsed from the date of seizure. I drew your attention at the time to the fact that the fingers of both these women were flexed into the palm of the hand through permanent contraction of the flexors, and I told you then, as it has since unfortunately happened, that they would never be able to use their hands; that the extensors would never regain the power they had lost, that the hand would always look like claws, and the power of motion in the upper limb would be almost completely abolished.

This is another fact taught by clinical experience which you should not ignore,[1] because you must not hold out the promise of

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a gentleman between fifty and sixty years of age, was seized with complete right hemiplegia, but in a short time regained some power over his extremities. The curious circumstance was that he could scarcely walk, when he had almost perfect use of his arm, and could even write and shave. Within a few months, however, the leg got worse, the arm itself lost power, and worse than all, his intellect failed perceptibly. In less than a year from the date of seizure, he died completely demented.—Ed.]

[1] [If the deformity of the hand be the result of atrophy and degeneration of the extensor muscles, the case is, no doubt, beyond all hopes of recovery, or even of improvement. But if it be seen at the very outset, when the contraction of the flexors is slightly marked, and there is only a difficulty in fully stretching out the fingers, faradization of the extensors, according to the principles laid down by Dr. Duchenne (de Boulogne), in his "Traité de l'Electrisation localisée," will sometimes, I believe, succeed in arresting the deformity.

In a case lately under my care at the National Hospital for Paralysis and Epilepsy this good result was obtained.

S— S—, a cabman, aged 33, married; a stout, strongly-built, middle-sized man. His previous health has been excellent, he has drunk pretty freely, but never to excess. On February 5th, 1865, he applied as an out-patient to the hospital with incomplete right hemiplegia.

A month before, whilst apparently enjoying the most perfect health, and without any premonitory symptoms, he found, on getting up in the morning, that his right arm and leg were numb and weak, and that his speech was thick and embarrassed. His wife noticed that his face was drawn to one side. He had great difficulty in dressing himself, but nevertheless went out as usual and drove his cab. In the course of the afternoon the weakness and numbness of his right side increasing considerably, and giddiness supervening with some mental confusion, he went home and took to his bed. He maintains that he never lost consciousness, and that he passed a quiet night. The following morning he had entirely lost all power over his right side, and could not utter a single word. For a week or ten days the paralysis remained complete, and the inability to speak persisted. At the end of that time, some degree of power returned in the limbs, and he could speak

cure, or even of improvement, in such cases, as the symptoms, far from getting better, will grow worse with every succeeding year.

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again, although with a thick voice. At the end of three weeks he could manage to walk about with the help of a stick.

When he came to the hospital there was imperfect right hemiplegia, the leg being considerably less affected than the arm. The fingers of the right hand and the thumb were turned into the palm, and the patient could not extend them in the least. The arm itself could not be raised higher than the chin. Numbness was complained of, but no appreciable diminution of tactile sense could be detected. The sensibility to differences of temperature, to pain, and tickling was perfect. The left angle of the mouth was on a higher level than the right, and there was slight deviation of the apex of the tongue to the right. Speech was natural.

Iodide of potassium, in two-grain doses, three times a day, was prescribed internally; and the extensors of the hand and the deltoid were galvanized three times a week by Mr. Radcliffe, the medical superintendent of the hospital. Within a fortnight a sensible improvement took place, which went on increasing by degrees. At the present time (April 15, 1865) the patient can move his arm perfectly, stretch out his fingers fully, and use his hand for writing. He no longer drags his leg when walking, and he can stand on it alone. All trace of paralysis of the face has completely disappeared. —Ed.]

LECTURE II.

ON APOPLECTIFORM CEREBRAL CONGESTION, AND ITS RELATIONS TO EPILEPSY AND ECLAMPSIA.

§ 1.—The existence of Cerebral Congestion is not contested; but it has been singularly abused, in order to explain cerebral phenomena in the production of which Congestion plays no part whatever.—Sudden and transient fits of Apoplexy are among these, and the so-called Apoplectiform Cerebral Congestions are oftener connected with Epilepsy than is generally believed.—A few considerations on the sudden and irresistible impulses of Epileptics in general, and on the inferences to be drawn from them in a medico-legal point of view.

Gentlemen,—*Apoplectiform Cerebral Congestion* is a term usually applied to a group of transient phenomena occurring suddenly, and resembling those of apoplexy properly so called. These latter are well defined in the aphorism of Boerhaave, which I have already quoted in the preceding lecture; namely,—"*Apoplexia dicitur adesse, quandò repentè actio quinque sensuum externorum, tùm internorum, omnesque motus voluntarii abolentur, superstite pulsu plerùmque forti, et respiratione difficili, magna, stertente, unà cum imagine profundi perpetuique somni.*"

When these apoplectic phenonema are transitory, the case is said to be one of *apoplectiform cerebral congestion;* when they are persistent, cerebral hæmorrhage is, in the majority of cases, supposed to have taken place to a large amount. It is a current opinion, as you are well aware, that *apoplectiform cerebral congestion* is a common complaint, and this opinion is so generally accepted, so firmly established, that it seems strange for any one to appear to doubt it. During the first years of my practice I saw, or thought I saw, a pretty large number of cases of apoplectiform congestion, but for a long time I have not seen any; yet other medical men see as many as before. Let us, therefore, inquire on whose side the error lies. A man, for instance, with or without premonitory symptoms, falls down suddenly in an apoplectic condition. When picked up he looks stupified, and for a quarter of an hour, an hour, or perhaps more, he complains of heaviness of the head and mental confusion, and staggers in walking. On the next day all these symptoms have disappeared. In such a case the patient is said to have had apoplectiform cerebral congestion. I used to say so like the rest, but I do not now.

Another man, whilst walking, is suddenly seized with giddiness. He loses his sight and the faculty of speech, merely muttering a few unintelligible words. He staggers, and sometimes falls down; but rises immediately. The whole set of symptoms have occurred within a few seconds, and are followed by a slight heaviness of the head only, and sometimes by transient mental confusion; but after three or four minutes he is as well as before. Such a case is said to be one of slight cerebral congestion. I also used to say so, but no longer say so now.

Why, then, have I altered my views, gentlemen? Not, certainly, from a love of paradox; but because facts have forced on me a new conviction. In the year 1845 a friend of mine was found in his bed in a state of insensibility. His face was turgid and livid, his intellect in abeyance, and all power of motion and sensation completely lost; there was stertor also. He was a vigorous man, aged 42. How long he had been in this condition his wife could not tell; for she had been awakened by a strange snoring noise, and she had sent for me. I had already, at that time, given up bleeding in the treatment of apoplexy. I had the patient placed in a half-sitting posture, and threw cold water in his face. I also applied two ligatures round the upper part of his thighs, in order to retain temporarily a large quantity of venous blood in the vessels of the lower limbs, (although I in reality expected little from the measure), and I waited. An hour scarcely elapsed before the patient regained the power of motion and feeling, and answered questions pretty well to the point. On the following day, great lassitude was the only symptom remaining.

Some time afterwards I was fetched in haste to see a neighbour, aged 70, who had been seized with apoplexy on the Boulevards. He had been insensible for a quarter of an hour, but was recovering his senses as I arrived. He did not yet recognize me, however, and looked vacantly round, moving his arms and legs about, without being conscious of it. His lips and nose were swollen, his eyes injected. By degrees, and within a few hours, he recovered entirely, without my having had recourse to any active measures. His valet then informed me that his master had, in the last two or three years, had several attacks of the same kind, and that the symptoms had passed off in the same way, once after bleeding, and on the other occasions after a mustard foot-bath. In the same year I was consulted by a solicitor from the country, aged 35, who in the course of the previous six months had suffered from three apoplectic fits. He had been bled and purged on each occasion, to his great satisfaction, and leeches were applied once a month round his anus. The last attack had occurred as he was going up a staircase to his apartments, on his return from some important pleadings. His head had struck against the stairs, and

there were still on his forehead the marks of a pretty deep cut. The apoplectiform phenomena had lasted an hour at the most; and when I saw him, his intellect, sensibility, and power of motion were perfectly normal.

I can with difficulty believe that apoplexy occurs in persons aged 35, particularly when the attacks return every two months. It immediately occurred to me that the case was one of epilepsy, and I suggested it to the medical man who had sent the patient to me. His answer was that nothing authorized my suspicions, and that convulsions had never been noticed. I still maintained my opinion, however; and shortly afterwards the poor man had, in court, a regular epileptic fit, which unfortunately left no doubt in anybody's mind, and he was compelled to give up his profession.

But my attention had now been roused; I asked myself whether so many persons whom I had seen with apoplectiform cerebral congestion were not epileptics, and I kept on the watch. My first patient soon had other attacks, and he now has sometimes as many as four or five epileptic fits in a day, and very often the vertigo of petit-mal. He has lost his sight, and his mind is considerably impaired. As to the old man whose history I have related to you, he is still living, and has almost every year one or two similar attacks. Since the day he fell down on the Boulevards, he never goes out unless accompanied by a servant, who has informed me that his master makes grimaces when on the ground, and has startings in one of his arms, which last scarcely a minute, but are amply sufficient to characterize epilepsy.

Since that time, whenever I have been consulted for a case of apoplectiform cerebral congestion, I have inquired with the greatest care whether, from time to time, there were, during the day, sudden and transient attacks of vertigo, having the characters I have indicated above, and whether those congestive seizures occurred more frequently at night than in the daytime, and whether also there had been *nervous* twitches in the beginning of the attack. In every case, almost, when the seizures had occurred in the presence of witnesses, convulsions could be made out. When they had taken place in the night, and during sleep, I was told that the urine had been sometimes passed involuntarily, and that for a few days the tongue had been sore. The face, forehead, and neck had often been covered with small ecchymoses, looking like flea-bites. I was told particularly that the attacks recurred at pretty short intervals, and left no lasting traces. In a word, epilepsy became plain when it was sought for.

Not a month elapses without my seeing in my consulting-room patients suffering from epilepsy, who are said to have had apoplexy. Not a week, perhaps, goes by without my being con-

sulted by adults and old people, or for children, affected with epileptic vertigo, who are said to be suffering from slight cerebral congestions. Although epilepsy, in all its forms, is better known now than five-and-twenty or thirty years ago, yet many practitioners will not believe in so terrible a disease; and even if they recognize it, they will not tell the patient's friends the real nature of the case, and prefer to leave the painful task to the consulting physician.

Very frequently, epileptic vertigo gives rise to symptoms usually attributed to cerebral congestion; symptoms to which attention has long been drawn by those who specially devote themselves to the treatment of the insane.

After an attack of vertigo, the patient is frequently delirious for a few minutes, and perhaps longer. The records of courts of justice and of police-offices are full of cases of suicide and of murder too often attributed by medical men to what they call cerebral congestion, but which should be ascribed to epilepsy. It may be said, almost without fear of making a mistake, that if a man suddenly commits murder, without any previous intellectual disturbance—without having up to that time shown any symptoms of insanity, and if not under the influence of passion, or of alcohol, or any other poisonous substance which acts with energy on the nervous system, it may be said, I repeat, that the man is afflicted with epilepsy, and that he has had a fit, or, more usually, an attack of vertigo. The reason why these strange acts are attributed by most medical men and by magistrates to passing cerebral congestion, is that the epileptic seizure is sometimes mistaken, and that the vertigo is almost always so.

I never pretended, gentlemen, that because a culprit is epileptic he should be exonerated from all criminality. Let a barrister use this argument; let him pretend that his client was not a free agent at the moment when the criminal act was committed; I grant it, but I will never, for my part, dare support such a doctrine before a court of justice. I am perfectly convinced that many epileptics are great criminals in the moral sense of the word, and that the acts of which they are guilty have been premeditated, and committed by them as free agents. But in such cases, the preparations for, and the perpetration of the crime, are in nothing different from what usually happens. The epileptic, if not insane in the interval between his fits, is like any other man, and as such, is amenable to the laws. On this point no difference of opinion exists. But if this same individual has committed a murder, without any possible motive, without profit to himself or any other person, without premeditation or passion, openly, and consequently in a manner quite different from that in which crimes are usually committed, I have the right of affirming before a magistrate that the criminal impulse has been the result,

almost to a certainty, of the epileptic shock. I would say *almost* if I had not seen the fit; but if I myself, or others, had seen a fit or an attack of vertigo immediately precede the criminal act, I would then affirm most positively that the culprit had been driven to the crime by an irresistible impulse, and he would be absolved by virtue of Art. 64 of the Penal Code.

It would be a mistake to believe that epileptics have sudden and irresistible impulses in the interval, and independently of the fits. When insanity has been brought on by epilepsy, as is, unfortunately, very common; when acute mania follows, for a few days, on a convulsive fit, no doubt can exist; and persons so afflicted are rarely brought before a court of justice if they commit a crime or misdemeanour. Where dementia is evident, the law does not punish. The magistrate orders the man to be confined, because he owes protection to society that is menaced, and to the poor madman himself, who is legally incapable.

But *the epileptic shock can strike at the will.* The perfect intelligence of the epileptic immediately before and shortly after the attack, his absolute moral liberty in the interval between his fits, can alone make him appear guilty. Those, then, are the conditions which should be studied.

In general, the question of guilty or not guilty is not raised when the crime or misdemeanour has been committed immediately after a fit, when those who witnessed the crime also witnessed the convulsions, any more than it is in the case of a maniac confined in a lunatic asylum, or of an hospital patient under the influence of delirium, who may commit any acts of violence. It may happen, however, that the fit does not occur in presence of any witnesses, or that the acts which are committed soon after are not seen by those who witnessed the fit, and then doubts may arise.

The following case was related to me by Dr. Jozat :—A young man, whilst on his way to the Palais Royal, in company of some friends, with whom he was going to dine, suddenly falls down on the "Place Louvois," but soon gets up again, and rushes on the passers-by, striking them with violence. He is taken to the police-station, and for some time keeps insulting the soldiers who hold him, and spitting in their faces. Now, had there been no witnesses of the epileptic attack which had preceded this extraordinary scene, and had not the physician who related the fact to me, interfered, the young man would have been tried for rebellion against the police authorities. It will be easily conceived how difficult it is to arrive at the truth, when the epileptic and the victim of his violence have been quite alone. And on this point allow me to bring before you a certain number of cases that fell under my own observation, and for the truth of which I can vouch. I was very recently consulted by a newly-married

couple. The lady told me that, shortly after her marriage, she had been awakened one night by strange movements of her husband, who had suddenly struck her with awful violence. Had she not managed to ring the bell, she added, and a maid-servant rushed in and delivered her, she might have been seriously hurt. Another scene, of the same kind, had again taken place a few days before I was consulted; but on this occasion she awoke in time, lighted a candle, and saw the convulsions with which her husband was seized. Flight saved her from the violence which immediately followed. These details were told me in the presence of the poor man, who was perfectly conscious of having felt something that he could not account for, and who now informed me that he had often, before his marriage, had attacks of vertigo, the character of which had not been recognized by the medical men whom he had consulted.[1]

I have still in one of my wards at the Hôtel-Dieu a young girl, of a quiet and gentle disposition, who sometimes has, within the twenty-four hours, as many as a hundred attacks of petit-mal. On the night of her admission she was put in a separate room, with a very intelligent nurse. About the middle of the night, she got out of bed after an attack, and began to beat the nurse, who woke in a fright. Scarcely half a minute elapsed before the patient recovered her senses, and got into bed again, ignorant of what she had done. You have all heard, without doubt, of that highly intelligent lady, and perfectly respectable in every respect, who, in a drawing-room, at a theatre, in church, or when walking out, suddenly makes use of most insulting or obscene expressions, of which she is said to be unconscious.

I have myself known a very intelligent magistrate, of whom I shall speak again by-and-by, who was subject to frequent attacks of epileptic vertigo. His sister had been confined at Charenton,

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[1 A case very much like the above occurred in my own practice, in the autumn of 1864. I was sent for in haste one evening to a gentleman, who had consulted me some time previously for epileptic vertigo. I found him in a state of excitement, which subsided soon after my arrival. I learnt from his wife that they were sitting quietly by the fireside after dinner, when he suddenly stopped in the middle of a sentence, turned deadly pale, and fainted away. She rushed up to him to prevent him from falling, when he suddenly got up, looking wildly delirious, and struck her repeatedly. Had not the servants, alarmed by her cries, come in and held him off, she might have been very seriously injured. He was scarcely recovering himself when I entered, and he burst into tears on hearing the above details. When questioned, he positively declared that he was not only unconscious of what he was doing at the time, but had no recollection of it.

See another case of the kind quoted from Dr. B. A. Morel's "Treatise on Mental Diseases," in Dr. Forbes Winslow's work on "Obscure Diseases of the Brain and Mind," p. 390, 3rd ed.—Ed.]

where I knew her. He was president of a provincial tribunal. One day he gets up all of a sudden, mutters a few unintelligible words, and goes to the deliberating-room. He is followed by the usher, who sees him make water in a corner. A few minutes afterwards he returns to his seat, and again listens with intelligence and attention to the pleadings, momentarily interrupted. He had no recollection of the incredibly incongruous act which he had committed.

I could cite an endless number of similar instances, borrowed from my own practice and that of others; but I wish to answer one of the gravest objections made by medical men, and still more by magistrates, to the theory of sudden and irresistible impulses in some epileptics. The disturbance of the reason which follows a convulsive fit, and especially an attack of vertigo, is not always recognized so easily as it might be supposed. A medical man, for instance, is sent for to see an epileptic immediately after an attack. The patient answers questions pretty well to the point, follows out the doctor's prescriptions, takes a foot-bath, allows himself to be bled or leeched, and describes his feelings pretty accurately; but a few hours later he has not only forgotten what occurred during the attack, as the rule is, but he has even forgotten all the above circumstances, in which he had apparently concurred with so much presence of mind. It must, therefore, be concluded that his intellect had been deeply perturbed. Who, now, can calculate the degree of liberty possessed by a man in this state of transition between the actual attack and the complete recovery of the mental faculties? Is there a medical man bold enough to pronounce on this point, and to affirm that a crime committed after the attack must entail responsibility?

Not only, gentlemen, may the patient's reason remain in a perturbed condition for some time after the attack, although a superficial observer may not perceive it, but it sometimes happens that, during the attack, the epileptic seems to retain enough reason to appear free. Allow me to cite a few instances in illustration.

The young girl now in my ward, to whom I alluded just now, goes, during her attacks of vertigo, through certain acts that require, in some measure, liberty and intelligence. If, at the outset of the attack, any one snatches from her an object she is holding, she rushes on him to regain possession of it, pursues him without staggering, without stumbling or knocking against anything in her way, and is even violent if she be resisted; then, all of a sudden, before half a minute has elapsed, she exclaims, "*It is over,*" stops, and falls into a state of prostration. If questioned at once, she has no recollection whatever of what has just occurred. When I treat more particularly of epilepsy, I

shall detail to you the case of a young man, a great musical amateur and very skilful violinist, who is afflicted with epilepsy. He is so passionately fond of music that he plays second violin at some theatres without any remuneration. He has often been seized with vertigo whilst playing a piece; but during the attack, which does not last more than ten or fifteen seconds, he continues to play in perfect time. He then comes round, knows full well that he has had a fit of absence, and continues to play without difficulty.

The lady whom I mentioned just now as being liable to singular and irresistible impulses, prompting her to use, without her being conscious of it, most strange expressions, makes in a loud voice witty and pointed remarks, contrary to the rules of society. But, although she acts under the influence of an irresistible impulse, her remarks are so perfectly apposite, however, that persons, not familiar with the phenomena of epilepsy, must incline to believe that they are made intentionally. If, instead of insulting or obscene expressions, or epigrams, you substitute murder, say whether there would be crime, and whether, in such a case, the article 64 of the Penal Code would not find its application?[1]

The magistrate concerning whom I told you such a singular anecdote, remained for some time in a disturbed mental condition after an attack; but this state was noticed by his wife alone, who was an excellent judge of it, and watched him with great solicitude. He belonged to a literary society, which held its meetings at the Hôtel de Ville of Paris. At one of these, during a discussion on an important historical point, he is seized with vertigo. He runs quickly down to the Place de l'Hôtel de Ville, and walks about for a few minutes on the quays, avoiding with success both carriages and the passers-by. On recovering himself, he perceives that he has come out without his great coat and his hat, returns to the meeting, and resumes, with a perfectly lucid mind, the historical discussion in which he had already taken a very active part. He retained no recollection whatever of what occurred between the beginning of the attack and the moment he recovered himself.

Now had this patient quarrelled with and killed a man in the streets, would a magistrate have believed that an individual who, five minutes before and five minutes after, was remarkably intelligent, and who, during this pretended nervous seizure, seemed to have his free will, could commit murder under the influence of an irresistible impulse?

Every physician who has studied epileptic vertigo practically

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[1] [See an "Essay on the Responsibility of Epileptics." By Dr. Baillarger. Translated and published in the *Medical Critic and Psychological Journal*, edited by Dr. Forbes Winslow, vol. i. p. 507.—Ed.]

must have seen cases of individuals speaking and answering questions during the attack; speaking, it is true, in a strange, jerked voice, but still answering questions *to the point.* The paroxysm once over, they have no recollection of what has just passed.

I had a motive, gentlemen, for going into all these details, and you will soon see that they are the key to the solution of the question. I showed you by numerous instances in point that sudden and irresistible impulses are of usual occurrence after an attack of petit-mal, and pretty frequent after a regular convulsive fit. I stated that the patients should not be held responsible for their acts, whether these be followed or not by grave and painful consequences, the gravity of the act itself having nothing to do with the question. The individual is not a free agent for the time, and is, therefore, free from guilt. This is the first point. The next is, that the epileptic acts unconsciously and without retaining any recollection of what he has done.

The very reverse obtains in the case of an insane individual, who is prompted to his acts, it is true, by hallucinations or by motives connected with his delirium, but who still acts with a very determined will, after long and matured premeditation. He always knows what he has done, and is, therefore, conscious of his act; for if he commits the crime suddenly, and sometimes from an irresistible impulse, he does so, in most cases, under the influence of hallucinations which justify the act in his eyes. Whenever delirium supervenes in the course of an acute disease, whenever it constitutes what is, by common consent, termed insanity, or follows chronic poisoning by alcoholic drinks, or is the consequence of repeated attacks of epilepsy which lead to dementia, the acts prompted by it are voluntary, methodical, and the patients always remember them.

I admit that the acts of an individual poisoned by alcohol, belladonna, or hachisch, may be unpremeditated and committed under the influence of an irresistible impulse, and that all recollection of them may be completely lost, as in the case of an epileptic. I admit that an idiot whose intelligence and moral sense do not rise to the level of those of the lower animals, may kill a man as he breaks a piece of wood, without being conscious of his act, or keeping any recollection of it. But I never meant to include these particular cases in the general proposition I laid down, since I supposed a complete integrity of the *reason* immediately before and soon after the perpetration of the criminal act.

That proposition I maintain, therefore, and I do not see that the arguments opposed to it in the discussion at the Academy of Medicine have as yet refuted it.

I dare not here, I confess, raise the question of irresistible

impulses in hysterical and in pregnant women. On that point I deny and I affirm nothing, but remain very incredulous.

§ 2.—Apart from Epilepsy, a great many cases of so-called Cerebral Congestion, in what is popularly known as the *coup de sang* (*ictus sanguinis*), belong to the class of internal convulsions, of vertigo occurring in connection with disease of the internal ear, and with dyspepsia.—What happens in the brain in these attacks is much more nearly allied to syncope than to congestion.—The apoplectic stupor of Cerebral Hæmorrhage, of Epilepsy and Eclampsia, is due to what I have called "*cerebral surprise*."—Epilepsy and Eclampsia present remarkable analogies.—The condition of the cerebro-spinal axis, of which they are both an expression (a condition unknown in its essence), suffices for producing stupor.—The Cerebral Congestion, which in attacks of Epilepsy and Eclampsia may be pushed as far as hæmorrhage, is a secondary phenomenon.

But let us return to cerebral congestion. One reason why epilepsy is often unrecognized, is the repugnance felt by families to confide the sad complaint even to the physician. A mother may have witnessed a regular convulsive fit, and yet is unwilling to believe in epilepsy. When questioned by the physician, she will mention the loss of consciousness, the coma, but will often omit the convulsions. She will ask for remedies against the accidents which follow the attack, but will not allow the truth to be suspected. I have often been consulted by persons who were perfectly well aware that they were afflicted with epilepsy, but who only spoke of congestion. Wives conceal the nature of their husbands' complaint; husbands, of their wives' affliction; and in most cases parents hide the symptoms presented by their children.

The physician is therefore constantly deceived in cases of epilepsy; deceived by the patient who knows nothing of his attack, except that he lost his senses, and remained several hours in a state of semi-stupidity; and he is deceived by the parents, who are with difficulty persuaded to confess that a member of their family is an epileptic. He is misled also by what he was taught when a student, namely, that apoplectiform cerebral congestion is a common complaint. There need be no surprise then, that congestion is so generally accepted. Medical men themselves are often the authors or accomplices of these mistakes. One of my best friends was an epileptic. As the disease was hereditary in his family, his wife dreaded lest her only son should come in for the sad legacy, and the name alone of *epilepsy* inspired her with intense terror. When I first found out the painful truth, I confess I had not the courage to tell her of it. I spoke of cerebral congestion, and I succeeded in persuading herself, as well as her son and her intimate friends, that epilepsy had nothing to do with the terrible complaint he was suffering from.

A few years ago, under similar circumstances, I wilfully committed

the same error. A young lady, belonging to a family I knew intimately, had married a gentleman of good standing. A year after her marriage, she told me that she had fainted in the night, had passed her urine involuntarily, and had bitten her tongue. The next morning she had felt general lassitude, and had a violent headache on waking. Fortunately she did not sleep in the same bedroom with her husband. I confess that I had not the courage to tell her or her friends the awful truth. For several years the fits recurred during the night only, and in the daytime she had frequent attacks of vertigo. Whilst staying at the seaside, she was one day on the beach, bathing one of her children in less than two feet of water, when she was seized with a fit, and was drowned in less than two minutes. The newspapers spoke of it as of death caused by cerebral congestion, and I did nothing to correct the mistake.

There is, I admit, one form of convulsive epilepsy which may simulate cerebral congestion. In some cases, but very rarely, at the beginning of a fit, in the tonic stage, when the muscles of the chest are perfectly rigid, it happens that instead of lasting only from fifteen to thirty seconds, this tonic condition extends over two or three minutes, and the patients die of asphyxia, in the same way as patients afflicted with tetanus die in a paroxysm, or animals poisoned by strychnine, as so well shown by Ségalas. In such cases there occur no clonic convulsions, with which persons not belonging to the profession are most familiar. All the time the tonic condition lasts, the face is swollen, the blood-vessels of the neck look distended, almost knotty, and there is in reality intense congestion, but of a passive character, analogous to what takes place during an effort. Active congestion is however diagnosed, although there has been, after all, an attack of epilepsy or of eclampsia. Physicians who devote themselves specially to diseases of parturient women and of infants will no doubt remember such cases, and will probably share my opinion.

My regretted friend Dr. Ménière, physician to the Deaf and Dumb Institution of Paris, had long ago observed a good many cases in which an individual seized suddenly with vertigo, nausea, and vomiting, after walking as if he were intoxicated, fell down, got up with difficulty, and remained for a time pale, bathed in cold perspiration, almost in a state of syncope. On similar attacks recurring frequently, they were at first regarded as due to cerebral congestion, and were actively treated with bleeding, leeches, and purgatives; but their frequency by degrees compelled a modification of the diagnosis, and excited considerable anxiety in the patient.

In the immense majority of cases, individuals so afflicted soon complained of tinnitus aurium, and even of hardness of hearing, for

which they consulted Dr. Ménière. One or both ears were then found singularly affected, and Dr. Ménière was enabled to collect hundreds of cases showing that these pretended cerebral lesions were in reality affections of the auditory apparatus. He investigated this point with extreme care, and succeeded in finding out that the internal ear was the starting-point of the phenomena in question, and that disease of the semicircular canals was the cause of the vertigo, the sympathetic vomiting, the paralysis of the limbs, and the sudden loss of consciousness.

Vertigo, connected with gastric disorders, is another complaint constantly mistaken for cerebral congestion. This strange form of neurosis is characterized by the following symptoms:— On the patient moving suddenly in bed, he feels the bed turn and carry him round with it; if he gets up, and particularly, if he then looks up, the giddiness becomes much greater. He sees everything turn round, he staggers, and is sometimes unable to remain standing, whilst he has all the time unbearable sensations of nausea, and is very often actually sick.

These curious symptoms are attributed to a rush of blood to the head, and, let us confess it, most physicians hold that opinion. They bleed their patients, therefore, they cup and leech them, and prescribe mustard foot-baths, doing, in a word, all in their power to remove the pretended congestion, which their strange treatment merely aggravates. These attacks of vertigo are more allied to syncope, and are consequently the reverse of congestion. However incredible this may appear, it is no less true that too many physicians still fail in recognizing the tendency to syncope, and confound it with cerebral congestion.

There is a symptom, however, which often accompanies cerebral hæmorrhage, and which by all medical men is regarded as indicative of congestion.

Thus a man, in whom cerebral hæmorrhage takes place, sometimes becomes suddenly insensible, and this abolition of the intelligence and of the power of motion lasts from a few hours to several days. He then comes round again, with the exception of a trifling degree of hemiplegia, which slowly diminishes and finally passes off, after a period varying from a few weeks to several months. As the first symptoms set in almost with the rapidity of lightning, and as there seems to be no proportion between their gravity and the subsequent impairment of the intellectual faculties and the power of motion and sensation, it is said that the cerebral hæmorrhage has been attended with congestion, and that the congestion, an essentially transient phenomenon, has caused the *apoplectic* symptoms proper, and on disappearing, has left behind it hæmorrhage to a small amount, and trifling paralysis. I do not mean absolutely to deny

the existence of this congestion, and I even confess that I am inclined to admit it within a certain limitation. There is, however, another symptom to which sufficient importance has not been attached, so far as I know,—namely, a kind of stupor, like what follows on commotion, and to which I have given the name of *Cerebral surprise*. When the brain is suddenly torn or compressed, it bears such a grave lesion with an impatience which varies according to individuals, but which may be very considerable in some cases. Wounds of the brain give us an illustration of this. When a soldier, for instance, is wounded in the head by a ball, or when a man, in a brawl, is stabbed in the head, and the knife enters the brain, he drops as if knocked down by a blow from a stick; but by degrees, notwithstanding the intracranial effusions of blood, which are a consequence of the wound, and even notwithstanding the inflammatory congestion inseparable from a laceration of the tissues, the intellectual faculties, the power of motion and sensation, are sometimes recovered with extraordinary rapidity, and thus give the inexperienced surgeon hopes which are unfortunately never realized. What I have called *cerebral surprise* is this instantaneous stupor. However incorrect the term I use may be (and I would gladly give it up), the fact exists and cannot be denied.

Experiments on the lower animals give still more positive results. If, after trephining the skull of a dog or a rabbit, a small leaden ball be introduced, through an aperture in the dura mater, between the skull and the surface of the brain, symptoms of stupor are immediately manifested, which gradually pass off, and are succeeded by an amount of hemiplegia proportionate to the compression.

In this experiment no cerebral congestion can be appealed to, and it must be admitted that the brain is somehow surprised by an accident which is accompanied by a transient disturbance. Am I not authorized, then, to suppose that when blood is suddenly effused into the corpus striatum or the thalamus opticus, the immediate stupor which is ordinarily attributed to a simultaneous congestion can, in part at least, be due to *cerebral surprise?* Does it follow, gentlemen, that I absolutely deny the existence of cerebral congestion? No, indeed, I admit cerebral hyperæmia, for I should be insane if I were to deny it; but I maintain that what has been called *apoplectiform cerebral congestion* is, in the greater number of instances, a symptom of epilepsy or eclampsia, and, in some cases, of syncope. I maintain that very often simple epileptic vertigo, and vertigo connected with a disordered state of stomach or with diseases of the internal ear, are wrongly looked upon as cases of cerebral congestion.

If the propositions which I have attempted to prove be true, it will be conceded to me that we must less frequently have

recourse to revulsives and to antiphlogistic measures in our treatment of these cases of pretended cerebral congestion, and that we must seek for other indications more in conformity with the views that should be entertained of the various conditions too often confounded under the same denomination. You remember, gentlemen, what stormy discussions were excited, in the beginning of the year 1861, by the opinions I now express, and which I then communicated to the Academy of Medicine.—("Bulletins de l'Académie de Médecine," Paris, 1861, t. xxvi.)

I neither pretended that I had discovered something, nor did I mean to teach my colleagues that attacks of epilepsy and eclampsia were followed by apoplectic phenomena; this had been said at all times and by every one. I only stated, and attempted to prove a fact seen and recognized by some physicians; namely, that sudden apoplectic seizures were oftener than is generally believed, connected with a *fit of epilepsy or eclampsia*. I spoke, indeed, of *transient apoplectic phenomena* occurring in an individual enjoying excellent health, with or without the premonitory symptoms which precede an attack of *grand-mal*, and leaving him, shortly afterwards, in the same state as before the seizure.

To speak unreservedly, gentlemen, I must at once declare that, in my opinion, epilepsy and eclampsia are two identical neuroses, with regard to their symptomatic expression and their proximate cause. When treating of epilepsy, I will show you that an attack of eclampsia is exactly like one of epilepsy, and that no physician will ever be able to distinguish between convulsions occurring in a pregnant woman, long afflicted with epilepsy, and convulsions in a woman seized with eclampsia, at the beginning of labour. So much for the symptoms. Now, as to the proximate cause, I believe it to be identical in both affections. When epilepsy manifests itself by monthly attacks in an individual with a tubercular deposit in his brain, there are in the brain and spinal cord, apart from the deposit of tubercle, no appreciable lesions other than those which exist in the so-called idiopathic epilepsy.

On dissection, if we find a deposit of tubercle, a cancer, or a bony tumour, the rest of the brain presents merely the appearances of vascular congestion, met with in the case of a true epileptic who has died in a fit.

What inference must we draw from this? It is this, that if the tumour in the brain be the cause of the convulsions, it is not the proximate cause; this does now, and will probably always escape us.

Eclampsia occurring in a child who is cutting his teeth or has worms, or is suffering from scarlatinal dropsy, does not in the least differ as to the convulsions from an epileptic fit, and yet these two affections are widely distinct as to their nature. I

mean to say that the molecular condition of the brain and spinal cord is, perhaps, the same in both cases.

Allow me to explain myself.

When we see an individual who for twenty years has been subject to almost periodical fits, and yet manifests no signs of insanity or general paralysis, we say that he is suffering from *idiopathic epilepsy*.

If in the intervals between the attacks there be hemiplegia, violent headache, or exclusively nocturnal pain, we suspect the *epilepsy* to be *symptomatic* of a tumour in the brain, or of tertiary syphilis.

If the convulsive disorder occurs in a pregnant woman with albuminuria, or in an individual with scarlatinal dropsy, or suffering from lead-poisoning, we call it *eclampsia*.

We give the same name to the convulsions which, in children, so frequently announce the invasion of febrile exanthemata,—of variola, for instance,—and to those which supervene at the close of *cerebro-meningitis*, or what is termed *cerebral fever*.

If the epileptiform convulsion takes place in an individual who has just been bled, or in an animal who is left to die of hæmorrhage; or, again, if it occurs, as in that curious experiment of Brown-Séquard, after the section of a lateral half of the spinal cord, under the influence of certain kinds of external irritation,—we also call it *eclampsia*.

What is the relation, then, of eclampsia to epilepsy, and of epilepsy to eclampsia?

If we look at the *convulsive character* alone of the two affections, *symptomatic* or *idiopathic* epilepsy, to use the bad divisions generally accepted, is only *recurring eclampsia*, and eclampsia is merely accidental and transitory epilepsy.

Eclampsia has been said to differ from epilepsy in the continuity and the occasionally prolonged duration of the convulsions which it causes; but although there be some truth in this distinction, there yet occur cases of eclampsia in which there is but a single attack, and cases of epilepsy with continuous seizures.

Now, for an *organicist*,—and I confess that I am one, in this sense at least, that I do not conceive a functional lesion without a modification of the organ which discharges the function,—every case of epilepsy or of eclampsia must be *symptomatic*, either of a tumour, or of some form of poisoning, or of a peculiar state of the blood, or of some inappreciable organic condition, as happens in epilepsy proper, in eclampsia from worms, or eclampsia which follows on venesection or hæmorrhage to a large amount.

In medical language (which I do not defend, and which I only use from want of another, and in order to be better understood)

we accept the name of *eclampsia* for convulsions occurring in the course of the cerebral fever of children; and why should we refuse to give the same name to convulsions due to chronic cerebro-meningitis, which, according to Royer-Collard, Calmeil,[1] and many others, causes the general paralysis of the insane?

We give the name of *symptomatic epilepsy* to convulsions which are caused by worms, or which are due to tubercle or cancer of the brain; and why should we refuse the same appellation to the convulsions which occur at the onset of tubercular meningitis?

Let us be logical, therefore, and let us admit that all epileptiform convulsions, although depending on very variable causes, are apparently the expression of the same intimate modification. If we admit this, we shall better understand the relation of eclampsia and epilepsy to what is, by common consent, called *apoplectiform cerebral congestion*.

As I shall tell you by-and-by, during the tonic period of an epileptiform seizure, the glottis is closed, and the patient makes a supreme effort, during which the face, the vessels of the neck, and necessarily those of the brain, get congested. The cerebral congestion may in such cases, then, be considered as secondary and passive.

But is the profound bewilderment, gentlemen, which succeeds an attack of eclampsia or epilepsy merely an effect of this passive congestion? I confess that I do not believe it; for the sudden loss of consciousness which occurs at the beginning of an epileptic fit, and which is from the first accompanied by a deadly pallor,—as so well pointed out by Calmeil, in his excellent thesis on epilepsy,—is the sign of such a deep modification in the functions of the brain, and perhaps in its intimate structure, that the stupor sequential to the attack is more probably a result of this modification than of the secondary passive congestion.

Mark, indeed, that we cannot admit, as many physicians do, that the attack of eclampsia is the consequence of a primary congestion, when, on the one hand, we see that the severity of the fit is by no means proportionate to the degree of previous plethora, and that, on the other hand, epileptiform seizures which follow on a considerable loss of blood are as severe as those noticed under perfectly different circumstances. Add to this—as you will read in the "*Journal de Physiologie*" of Dr. Brown-Séquard—that, at the onset of an epileptic fit, the great nervous centres and the medulla oblongata of an animal subjected to experiment become paler, instead of presenting signs of congestion.

Hence it follows, that what we all call *apoplectiform cerebral congestion*, and the apoplectic phenomena which succeed epilepsy

[1] "De la Paralysie considérée chez les Aliénés." Paris, 1826. "Traité des Maladies inflammatoires du Cerveau." Paris, 1859. 2 vols. in 8vo.

or eclampsia, may be nothing more than a condition analogous to the *apoplectic stupor* which immediately follows on some severe cerebral disturbance, and which certainly occurs independently of all congestion. Some think it very natural that cerebral congestion should produce such grave phenomena. But see what occurs in a woman during labour. As the child's head is going to pass the inferior outlet of the pelvis and the external organs of generation, the woman often makes most violent efforts. Her face becomes blue, her lips and eyelids swell, her skin gets hot and bathed in perspiration, and there can be no doubt but that the sinuses of the dura mater, and the whole substance of the brain share in this congestion. Is it under such circumstances that women are seized with eclampsia? Ask accoucheurs, and they will tell you that eclampsia manifests itself often before all signs of labour have shown themselves, and, in most cases, when there have scarcely been slight uterine contractions, which do not even attract the notice of the patient.

There was, it is true, albumen in the urine; but what has albuminous urine to do with convulsions, when a rational explanation is sought for?

It seems that in such cases convulsions are excited by a sympathetic cause as slight as the scarcely-perceived sensations which arise from the presence of worms in the intestines.

Children affected with whooping-cough may have so many fits of coughing in rapid succession, that an intense degree of congestion is thereby brought on; so much so, indeed, that they may have hæmorrhage from the nose, that their face will remain persistently puffy, and ecchymoses will in some cases form beneath the eyelids. There can be no doubt about the brain participating in the congestion. The fit over, they remain for a while in a state of bewilderment; but can this be compared with the lightning-like suddenness of an attack of eclampsia, and the apoplectic phenomena which follow it?

Acrobats, who go through many of their performances with their head downwards, never suffer from anything like apoplectic stupor. The porters of the Halle, who all day long carry heavy burdens, and who, constantly making powerful efforts, get almost blue in the face, whilst the blood-vessels of the neck are turgid, and look like knotted cords, are never seized with sudden loss of consciousness or of muscular power, at the very moment when they are exerting themselves the most.

Let us admit, then, that so long as the blood is not altered in its intimate composition, and is not extravasated, it is not so injurious to our tissues as is commonly said; and that something more than a purely physical congestion is needed to produce the apoplectic phenomena which succeed epilepsy or eclampsia. I understand better the disturbance which follows on that special

and essentially vital molecular condition which is termed flux or inflammation.

There are, therefore, and I lay great stress on the point, two very distinct conditions in an attack of eclampsia, or of epilepsy, whether idiopathic or symptomatic: 1st. *A cerebro-spinal modification,* unknown in its essence and in its nature, which in a second abolishes all the manifestations of animal life. Of the two, this is by far the more important condition. 2nd. *A secondary cerebral congestion,* which, although less important, may in some extremely rare cases be carried so far as to produce subcutaneous ecchymoses, cerebral capillary hæmorrhage, and even meningeal hæmorrhage.

Apoplectiform cerebral congestion is a term which has, in my opinion, been wrongly applied to the state of stupor which succeeds the complicated disorders I have just alluded to; and this term has had an injurious influence on the treatment employed, and on the notion medical men have formed of the disease.

Without quarrelling about names, and about the ultimate alterations which characterize what physicians call *apoplectiform cerebral congestion,* there can be no difference of opinion concerning the phenomenon itself. It is a state of profound stupor, analogous to that noticed in cases of individuals struck down by apoplexy, and it is attended with apoplectic phenomena; its cause being in a great number of cases epilepsy, idiopathic or symptomatic, or eclampsia.

These explanations were necessary before I could lay down the following proposition: *The same cerebro-spinal modification which causes the fit of epilepsy or eclampsia, the insultus, the ictus epilepticus, is sufficient to produce the apoplectic stupor which follows it.*

In a child, suffering from cerebral fever, there doubtless is some stupor, but never to a considerable degree. Let an attack of eclampsia supervene, however, and in a minute, from a state of scarcely appreciable stupor, he gets into an apoplectic condition.

What applies to the acute cerebro-meningitis of children applies also to the general paralysis of the insane, which is probably nothing more, after all, than a symptom of chronic cerebro-meningitis. In the latter case, with the exception of delirium, and of some uncertainty in his speech and gait, which do not escape those familiar with the diseases of the insane, the patient apparently enjoys good health; but on his having an epileptiform seizure, he is struck down instantly, and passes into an apoplectic condition.

In neither case is the cerebro-meningeal inflammation the proximate cause of the convulsive and apoplectic attack; it is only an

indirect cause, the immediate one being the minute central modification which brought on the attack.

Hence it follows that the apoplectic condition so often observed in the course of the paralysis of the insane is dependent on eclampsia, just as the analogous condition which follows an epileptic fit is dependent on epilepsy.

Let us pause awhile, gentlemen, and ascertain how far we have got on with the discussion. I have proved that transitory apoplectic phenomena occurring in an individual, in good health, and leaving him in the same condition after as before the attack, were, in almost every case, associated with epilepsy or eclampsia.

I have just shown that in cases of acute or chronic inflammation of the brain, and even in cases where the nervous symptoms arise merely from sympathy, as in typhoid fever, and in pneumonia, for instance, sudden apoplectic phenomena were almost always preceded by epileptiform convulsive phenomena. I can therefore repeat what I stated just now, namely, that the same modification of the nervous centres which produces the convulsions, is sufficient to account for the apoplectic stupor, and that the pre-existing inflammatory congestion is by no means the cause of the new symptoms that set in suddenly.

I am accused of making light of cerebral congestion, and of too easily doing away with it in Nosology. This is far from true, gentlemen, for I do not deny the existence of cerebral congestion, but only of that congestion which is said to produce *sudden and transient apoplectiform phenomena.* I admit determination of blood to the brain, as to any other organ, from irritation or inflammation. I admit that congestion evidently accompanies it, and that it is sometimes carried to such a degree that symptoms of apoplexy may be produced; but those symptoms are neither *sudden* nor *transitory.* Again I repeat, I only meant to speak of sudden and passing apoplectic phenomena, and, as far as they are concerned, I maintain my first opinion. If I make light of cerebral congestion, and refuse to see it where others do, you will agree with me that it was formerly, and is still, too lightly accepted.

Hemicrania and simple headache are said to be due to cerebral congestion. The stupor of typhoid fever, of typhus, pneumonia, the plague, variola, scarlatina, is set down to the account of congestion; and so is the delirium of pneumonia, of hysteria, St. Vitus's dance, erysipelas, &c.

Sleep itself has by some physiologists and physicians been ascribed to cerebral congestion. Therefore, whenever stupor and drowsiness showed themselves, whenever delirium or a tendency to dreaming set in, cerebral congestion was admitted with a facility which now appears strange to most practitioners. Nobody knows what sleep is; and the resemblance between two individuals, one of whom is plunged in a deep sleep after great

fatigue, and the other after an attack of apoplexy, has probably led medical men to attribute to one and the same cause conditions which have but a deceptive resemblance.

This singular opinion, however, which was not based on experiments, has strangely influenced the notion formed of the action of poisons.

If opium induced sleep, it was by causing cerebral congestion. Solanaceous plants, ranunculus, colchicum, digitalis, prussic acid, &c., caused stupor, because they induced cerebral congestion. The same obtained with *viruses* and with animal poisons, whether wholly produced in the living organism, in the course of toxæmic diseases, or whether introduced from without. Profound stupor was always ascribed to congestion. I have already said how innocuous I believed congestion to be; besides, there is no need whatever to have recourse to congestion in order to explain the action of poisons. They are absorbed and circulate in the blood, and, therefore, come in contact with all parts of the system, disturbing them more or less completely, independently of the liquid which acts as their vehicle; and often, as shown by the experiments of Magendie, in an inverse ratio to the amount of blood accumulated in the brain, for example.

Excuse me, gentlemen, for having dwelt so long on this point. The opinion I expressed before you at the beginning of this conference seemed extraordinary at first, but I am sure that it no longer seems so to you now, and that you are convinced, as I am myself, that *sudden and transient symptoms* of *apoplexy are in most cases associated with epilepsy or eclampsia.*[1]

[1 Consult on this important subject Dr. J. P. Falret's work: "Des Maladies Mentales," p. 477 and following. Dr. Falret, although admitting that apoplectiform cerebral congestion may occur spontaneously, as well as in the course of various cerebral affections, concurs however in Professor Trousseau's statement that it may be one of the manifestations of epilepsy. The chief circumstances which, in his opinion, should be taken into account before deciding on the nature of the case are:—First, the age of the patient, as epilepsy generally occurs for the first time in infancy or in adult age. Second, the presence or absence of hereditary predisposition to epilepsy. Third, the exciting cause of the attack, *e.g.*, fear, or some violent emotion. Fourth, the previous existence of vertigo, or regular convulsive seizures. Fifth, biting of the tongue, and involuntary passage of the urine. Sixth, the instantaneous production of the attack, and the intermittent character of the seizures. The diagnosis, he adds, cannot be made from the characters presented by the attack itself, but from the other concomitant symptoms, from the circumstances which have preceded, which accompany and follow the attacks, and especially from the progress of the disease.—Dr. Falret takes pains to prove, in opposition, as he believes, to Professor Trousseau, that apoplectiform cerebral congestion may occur in the course of certain cerebral affections, as in cases of the general paralysis of the insane, tumours of the brain, &c. &c. But on reading the above lecture it will be clearly seen that Professor Trousseau holds the very same opinions, and that his views must therefore have been misapprehended by Dr. Falret, or have been modified since the discussion on this subject at the Académie de Médecine.—Ed.]

LECTURE III.

ON EPILEPSY.

Cases of Epilepsy.—Description of a fit.—How to recognize the feigned disease —Three stages: tonic convulsions, clonic convulsions, and stupor.—Synonyms: morbus major, morbus comitialis, morbus herculeus, falling sickness, haut-mal, &c. &c.—Sequelæ: sub-cutaneous ecchymoses, cerebral hæmorrhages, &c.—Cerebral and spinal lesions are effects, not a cause of epilepsy.—Exciting causes.—Status epilepticus.—Petit-mal.

Gentlemen,—We have lately had in our clinical wards several patients afflicted with epilepsy. One of them was a young man, aged 18, who occupied bed 18 in St. Agnes ward, and presented that peculiar form of the disease which has been called *partial epilepsy*. It consisted, in his case, of convulsions of the facial muscles, exclusively limited to the left side, and unaccompanied by any phenomenon usually met with in an attack of *haut-mal*, or by loss of consciousness. On inquiring into his previous history, we learnt that the disease first set in about six years ago, with attacks of *haut-mal*. These were very violent at the commencement, but gradually became less so, and although there occurred convulsions from time to time, he generally suffered from epileptic vertigo only.

Before proceeding further, let me call your attention to this *transformation* of epilepsy, a fact pointed out long ago by the practitioners who specially investigated the question; by Calmeil among others. Let me remark, however, that they spoke of the transformation of *petit-mal* into *grand-mal*, whilst in the case of our patient, the reverse occurred, the convulsions having preceded the vertigo.

You may remember, also, another of our patients, an American, who, after having tried the public institutions of his native country, obtained admission into different Paris hospitals, and finally went to, and, as I have been told, died in London. He was tall and powerfully made, and had been nick-named *the blue man*, because of the slate-blue discoloration of his skin, due to a prolonged treatment with nitrate of silver, to which he had been subjected in the United States.

You saw him in several of his fits. On a sudden, he shrieked out, struggled, and turned round on his own axis, catching hold of the bedstead when he could, and losing consciousness entirely. The fit lasted a few seconds, after which the poor fellow recovered himself, although for several hours afterwards he remained in a state of bewilderment, and almost stupefied. You remember the

fixed idea he had: he had heard that castration had been performed for the cure of epilepsy, and not a day passed but he begged to be operated on. It was only after he became convinced of our determination not to accede to his request, that he left the hospital, and soon afterwards quitted France.

About the same period I had a third patient in bed 20, St. Agnes ward, whose history deserves to be related in detail. He was 36 years old, and had specially come from Bouconville (in the department of Ardennes) to be treated in Paris.

He had the aspect of a man of a robust constitution, and he stated that, indeed, he had never been ill. For four years and a half he had served, as a marine in Guadaloupe, and had enjoyed excellent health there. The only ailment he ever had was *chronic coryza*, dating many years back, and which ceased suddenly at the time when he first became subject to attacks of *haut-mal*. This coincidence led him to ascribe his disease to the sudden disappearance of the coryza. He affirmed that he had never been addicted to spirituous liquors. None of his relatives, direct or collateral, had ever suffered from nervous disorders; and his own child, then four years old, was in excellent health, and had never had convulsions.

The disease dated five years back. One night he had been suddenly awakened and frightened by horrible shrieks from his wife, and a few days afterwards he had his first attack.

In the beginning, these seizures were characterized by a sensation of inward cold, of rigors, and, to use his own words, of trembling, seated sometimes in the arms, the legs, or thighs, and sometimes in the pit of the stomach, or various parts of the body. This sensation spread all over him, and lasted a few minutes, without being attended with loss of consciousness. The attacks recurred at irregular intervals, rarely longer than four or five days, and were brought on by the slightest painful emotion, the least variation of temperature, a draught of cold air, or exposure to a hot sun. They gradually increased in severity, and within the last few months had become considerably more frequent and violent. They were now regular convulsive seizures, similar to those he had on admission, and several of which we witnessed ourselves. On the day of his admission, he had just lain down, when he suddenly got up, taking hold of the bar across his tester-bed, then throwing his arms about, began to vociferate in the most atrocious manner. His face was of a purple-red colour, his looks haggard, his voice loud, and his articulation rapid. He looked exactly like a delirious maniac. The attack had set in with quivering of the legs, followed by convulsions. He was so wildly delirious, that he frightened the patients in the ward. He had rushed out of bed, and had to be confined in bed with a strait waistcoat. He was perfectly unconscious of his acts,

and kept insulting those who were attending him. This fit lasted about twenty minutes, and without any transition he became calm. He spoke distinctly, and begged to be unloosed, as he felt the fit was over. I shall again call your attention presently to these phenomena of furor, and I will point out to you their medico-legal importance in determining the degree of moral liberty enjoyed by some persons, who, without any motive, have suddenly committed acts of violence, and even murder.

On the day following, the patient related to us his previous history, adding that within the last few months only had his fits been accompanied with loss of consciousness. Once his wife, on returning home, was surprised to find blood on the floor of the room; he was astonished himself, and on putting his hand to his head he felt a wound which he had received on falling down during a fit, of which he had no recollection.

His fits were generally preceded by the sensations I have already described; he next lost consciousness, was convulsed, and immediately became delirious. The attack lasted from twenty minutes to an hour even. He then became calm again, but complained of general lassitude, and usually of headache, which he compared to the compression that would be produced by a circle of iron. He was oftener seized at night than in the daytime. Of late his memory had seemed to fail; sometimes he felt confused and had a difficulty in collecting and in expressing his thoughts. He had become impotent also.

During his stay in the ward I had an opportunity of having him watched carefully, and of observing myself what happened during his fits. They never occurred in the same way. Once he was seized when walking out in the garden, and a companion who was with him thus related the circumstances :—

He turned pale suddenly, in the midst of a conversation, looking haggard, with his teeth chattering and his arms moving about in a disorderly manner. He was made to sit down on a bench, and his face then growing red, he laid hold of his companion's coat, as if he wished to strip him, and when asked what he meant to do, answered that his companion ought to take it off. He spoke distinctly, and yet he was so restless that he could with difficulty be kept on his seat. This attack lasted ten minutes, and was followed by a condition of bewilderment and perfect stupidity. When made to go up to the ward, he offered no resistance, his gait resembling that of a man under the influence of liquor. On recovering himself, he remembered nothing of what had occurred.

On another occasion, I had just been talking to him. He was sitting on a chair at the foot of his bed, when I suddenly saw him beating the ground with his feet. His face was excessively pale, his features distorted, his look haggard. He kept nervously

looking about everywhere, under his sheets and under his own clothes, exclaiming, "Where is it? . . . . my spoon? . . ." I vainly tried to question him; he made no answer, and seemed unconscious of all that passed around him. He yet pushed away my hand when I touched him. This time he had no convulsions. The fit lasted two or three minutes, and left him in a state of prostration.

These cases, gentlemen, may have appeared very singular and exceptional to some among you, but they are met with pretty frequently, however. I must therefore call your attention particularly to them. In all these three cases, as in others you have also seen in my ward, epilepsy was the disease under which the patients laboured.

This is a very important subject, and I intend to investigate it with you. By pointing out to you the various forms which it assumes, I will try and enable you to recognize this disease, one of the most formidable which afflict mankind, by means of imperfectly developed, nay, apparently insignificant symptoms.

The term *epilepsy* conveys to non-professional persons, and we must confess it, to many medical men also, the notion of a disease characterized by convulsive attacks, generally of short duration, and attended with loss of consciousness, swelling of the face, distortion of the mouth and eyes, immobility of the pupils, and a good deal of foam at the mouth, tinged red with blood.

Such, in fact, is the definition, very imperfect though it be, of an *epileptic fit*.

But this is only one of the forms of epilepsy, and there are many others besides, which are perhaps more frequently met with, and which, however different they may appear at first sight, present the greatest analogies between one another. And I hope to be able to prove to you that they are, after all, the expression of one and the same disease.

The convulsive form itself is often mistaken, or rather confounded with other convulsive affections, such as hysteria, and particularly the various kinds of eclampsia. These latter, it is true, simulate epilepsy very closely, but are nevertheless perfectly distinct affections.

But first, how are you to recognize *real* from *feigned* epilepsy?

Thus army doctors will tell you that individuals often feign epilepsy in order to be exempted from military service. But the *real* disease is characterized by certain phenomena which do not escape the observation of an experienced practitioner, and could only be feigned by individuals thoroughly familiar with them. Esquirol, however, believed that even such persons could not perfectly imitate the disease. Yet he was deceived himself, and on this occasion:—One day, Dr. Calmeil and I were talking with

him on this very subject at the Asylum of Charenton, when suddenly Dr. Calmeil fell down on the floor in violent convulsions. After examining him for a moment, Esquirol turned round to me, exclaiming, "Poor fellow, he is epileptic!" But he had no sooner said so than Dr. Calmeil got up and asked him whether he still persisted in thinking that epilepsy could not be feigned. Although Esquirol made a mistake in this case, I still maintain his proposition, and I believe that even a physician, thoroughly familiar with all that takes place during a fit, will only imitate it imperfectly, because there are some phenomena which cannot be produced at will, as I will show you as I proceed.

Now let us see what usually happens during a fit.

All of a sudden, without any premonitory symptom, the patient utters a loud scream, and falls usually on his face. This is already an important fact, and characteristic of the *real* disease. A man who feigns epilepsy takes good care not to throw himself down in that way, or if he does so, he keeps his hands in front of him, in order to protect himself on falling. The true epileptic is thrown down with such violence, that his head knocks against any obstacle in the way. Sometimes he falls backwards, or on one flank, but in most cases, I repeat, he falls forwards, and it is, therefore, on his nose principally, his forehead, his chin, his cheeks—in a word, on the prominent portions of his face, that you will find either actual wounds or scars of old ones. Fractures of the skull, or of the bones of the extremities, dislocations, may also be caused by the fall. In some cases the patient falls into the fire and burns himself fearfully; instances even have occurred of persons found burnt to death, after falling into the fire, and whose faces were so charred as to be no longer recognizable.

When down on the floor, the patient presents symptoms which should be carefully studied, because, although they do not last long, they are yet very characteristic. As he falls down, the epileptic is not red, as it has been wrongly stated, but deadly *pale;* and this is another phenomenon which is necessarily absent in feigned epilepsy. Convulsions then begin immediately. They are tonic at first, consisting in a powerful contraction of the muscles, which are in a state of violent tension, without alternate relaxation. They are more marked on one side than on the other, a character of great value in an epileptic fit, because rarely absent. Sometimes even they are limited exclusively to one half of the body. You will see, for instance, one arm twisted on itself and drawn backwards, the hand flexed, the thumb forcibly adducted and hidden by the fingers, which are bent over it into the palm. The lower extremity is also convulsed: the foot is arched and extremely tense; the leg is forcibly extended and twisted on itself. The muscular rigidity is not to be overcome, and although

they contract convulsively with a certain degree of slowness, the muscles are agitated by quivering of their fibrillæ, which can be easily felt.

To the hand they feel as hard as iron. The twisting and forcible pronation of the limbs are so violent, that injuries may result; and I recently saw a case of spontaneous dislocation of the shoulder, which had not occurred at the time of falling.

Such injuries may even be inflicted in nocturnal attacks, occurring during sleep, and I shall by-and-by dwell on their significance, as regards diagnosis. The following is an instance in point:—

At the end of the year 1862 I was consulted by a gentleman, aged 50, who told me that he awoke one morning complaining of a sense of fatigue and of pain in the right shoulder, which was so acute as to completely prevent him from moving his arm. He had formerly suffered from acute articular rheumatism, and the medical man whom he sent for, after examining the painful joint, came to the same conclusion as himself, namely that it was affected with rheumatism. The pain in the joint and its extreme rigidity persisting, however, without abatement, the patient, after several months had elapsed, returned to Paris, and consulted Mr. Maisonneuve, who recognized a dislocation, which was reduced with great difficulty on account of its ancient date. Some time afterwards the same accident occurred again under identical circumstances, but on this occasion the dislocation was immediately reduced.

Certainly, gentlemen, no dislocation of the shoulder ever occurs in ordinary sleep, and after the patient had related to me what had happened to him on these two occasions, I did not for a moment hesitate to ascribe the dislocation to nocturnal attacks of epilepsy. Other details, told me by the gentleman himself, confirmed my diagnosis. He had, indeed, on several occasions since, suffered from sudden fainting fits, and from vertigo, about the nature of which no doubt could be entertained.

Allow me to revert for a moment to the peculiar circumstance that tonic convulsions, in an epileptic fit, are generally more marked on one side, and sometimes even exclusively limited to one half of the body. Those who feign attacks are not aware of this, and think they ought to be convulsed on both sides, although if they knew the circumstance they might imitate it. The muscles of the trunk are affected as well as those of the limbs. The sterno-cleido-mastoid, for instance, is thrown into contraction, and as a consequence, the head of the patient is drawn down to the shoulder on the affected side, and the face turned to the opposite side. This is another circumstance not known to impostors. The muscles of the thorax and abdomen are likewise in a state of tetanic rigidity, and the respiratory movements are

completely arrested. The fibrillary quivering I mentioned just now as being felt on laying one's hand on the chest of the epileptic, is no longer perceived. After these tonic contractions have lasted a few seconds, and the thorax remained perfectly motionless, the face then begins to redden, and it is then and then only, and not when the individual falls, that the veins of the neck get distended, and that the face turns livid, remaining so for a pretty long time.

At the time, however, when tonic convulsions affected the muscles of the limbs and trunk, the face was distorted from the convulsions of its muscles. The tongue also, violently thrust forward from the involuntary contraction of the genio-hyo-glossi, protruded through the half-opened jaws, swollen out and purplish, but not yet cut or wounded by the teeth, as it often is in a later stage. In some cases, however, even in this first stage, the tongue is caught between the teeth, and deeply bitten, when the mouth closes slowly, after having been hideously distorted and partially opened.

This may be termed the *first stage* of an epileptic fit, or *stage of tonic convulsions*. It lasts from ten to forty seconds at most, and the *second stage*, or that of *clonic convulsions*, then begins. The limbs are alternately flexed and extended, and it is this stage which characterizes the epileptic fit with which everybody is familiar, and which is easily simulated. It lasts from half a minute to two minutes at the most, so that the whole duration of the attack varies from two to three minutes, and in most cases, still less than this. Those of you who have witnessed epileptic fits may probably think that I limit the time too much, but it is only because three minutes of such a horrible spectacle as that of a man in a fit seem very long indeed, and appear to last three or four times longer than they really do. But observations made watch in hand, testify to the correctness of my statement, and indeed Dr. Calmeil has himself pointed the fact out, and laid it down as a general law.

The clonic convulsions are more violent on the same side as those of the tonic kind were. They come on at first every second, and sometimes at still shorter intervals. They affect the muscles of the face, as well as those of the limbs and trunk; and from the exaggerated contractions of the muscles of the chest which modify the respiratory movements, breathing becomes jerking and noisy.

The convulsive movements describe a gradually larger and larger circle, until at last the muscles are fully stretched out and extended suddenly, when the patient draws a deep sigh, and the fit is over; at least, the convulsions are over, for a third stage now begins.

In most cases, it is in the second stage that the tongue is

wounded: thrust forward through the half-opened jaws by the contraction of its extrinsic muscles, it gets squeezed and bitten by the teeth when the muscles which elevate and depress the lower jaw are thrown into clonic convulsions. The wounds which are thus produced, account for the more or less abundant hæmorrhage, and the reddened foam noticed in a great many cases. The blood may also come from the nostrils, or be poured out from the gums which are bruised through the breaking of one or several teeth occurring at the time of the fall, or during the fit itself.

With the clonic convulsions ends the convulsive attack proper; but the patient then falls into an apoplectiform condition, and looks like an animal that has been felled, or an individual in whom there has occurred a considerable extravasation of blood into the brain, or who is stupefied by drink. His breathing is stertorous, and during expiration his half-opened lips give issue to frothy saliva, which is tinged with blood. For a length of time varying from a few minutes to half an hour, he remains in this condition of profound stupor and complete immobility. His intellectual faculties and power of feeling are entirely abolished during and immediately after the attack, so that he may be pinched, pricked, or burnt, without being conscious of it. In those cases, which are unfortunately not uncommon, when the patient falls into the fire, he may be burnt in a most awful manner without expressing or feeling the slightest pain. On lifting his upper lid, his pupil may be seen to be dilated, and refuses to contract under the stimulus of the brightest light. He neither hears nor smells, and a bottle of strong ammonia may with impunity be held under his nose. These again are facts which cannot be simulated by impostors.[1]

At length the patient opens his eyes: at first he looks around him in a stupid, confused manner. If he be still lying on the

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[1 With regard to this "insusceptibility of the pupil when the sun or candle-light is thrown upon it," Romberg states that he knows no sign "so trustworthy for distinguishing between feigned and genuine epilepsy. At the same time, he adds, we must be careful not to confound the reflex sensibility which continues even during the paroxysm, with the cessation of cerebral sensibility, and we must guard against mistaking the impression produced by sprinkling water, or by applying a feather to the eyelid, for an act of consciousness."—Romberg, "Manual of Nervous Diseases," translated by Dr. Sieveking, vol. ii. p. 215.

Marc, in art. "Epilepsie simulée," in "*Dictionnaire des Sciences Médicales*," vol. xii. p. 542, mentions that "in cases of pseudo-epileptic seizures, the thumbs are more easily opened, and the subject immediately closes them again, because he considers it an essential feature of the disease. If during a true epileptic fit, on the contrary, we forcibly unbend the thumbs, which are drawn across the palm of the hand, they remain so till the end of the attack or close again on the supervention of a fresh convulsion."—Ed.]

ground, he attempts to get up; but his movements resemble those of a drunken man; he looks ashamed, and tries to avoid the observation of lookers-on. If questioned, he falters out a few unintelligible words, and he can scarcely give the simplest information concerning himself, such as giving his own name and address, or he even makes no answer at all. He allows himself to be led about, however, to be put inside a carriage and taken home without offering any resistance, but at the same time with as complete an indifference as if he was not conscious of what was going on.

For a few hours afterwards, or a day, a couple of days sometimes, he complains of headache, and of some mental confusion, particularly of some failure of memory. Sometimes, also, he remains temporarily paralyzed on one half of the body. But in general, by the next day, he has recovered his usual condition.

This is what is termed an *epileptic fit*, gentlemen, the *grand mal* or *morbus major* of Celsus, which authors have designated by other names, such as *morbus sonticus* (the fatal disease), *morbus lunaticus astralis*, so called because the motions of the stars, of the moon in particular, were said to influence the attacks; *morbus caducus* (falling sickness); *morbus comitialis*, because if a man were seized with epilepsy during a meeting in the forum, at Rome, the assembly was broken up; *morbus herculeus, heracleus*, so called because Hercules was said to have been an epileptic; *morbus sacer, divus*, because sent by the gods; *St. John's complaint, St. Giles's complaint*, as it was termed in the Middle Ages, and as it is still called in some departments in the south of France; and again, *morbus demoniacus*, at the time when epileptics were believed to be possessed with the devil. All these names are applied to the convulsive fit, or *haut mal*, the most striking and the most familiarly known form of epilepsy. But what everybody does not know, and what must be consequently pointed out, is the fact that *epileptic seizures* very often, in the beginning especially, *occur during the night;* and that an individual may thus be afflicted for eight or ten years, although nobody, not even himself, suspects the existence of this dreadful disease. Certain phenomena, however, and certain accidents enable one to recognize a past attack; such as contusions, and injuries of a more or less serious nature, inflicted on the patient as he falls down, or caused by the severity of the convulsions, of which he bears traces at least on some part of his body. Dislocations of the lower jaw, of which there are instances on record, and the mechanism of which is plain, dislocations of the shoulder, although rare, but of which I quoted an instance myself, point in the same way.

Even apart from these accidents, there are other circumstances

more frequently met with, and which have, on the whole, an important significance.

In the beginning of the year 1863, Drs. Tardieu, Legrand du Saule, and Caffe, were called upon by a court of justice to report on the mental condition of a lady whose interdiction was applied for. Their inquiries had for a long time remained fruitless, and although they had ascertained a certain degree of failure of memory, they yet could not call it dementia, and they felt great embarrassment at giving a categorical opinion, when they were informed that the lady sometimes suffered from incontinence of urine, both by day and by night. Now, indeed, was light thrown on the subject, and on questioning the lady more closely it became evident that she frequently had nocturnal fits of epilepsy, during which her urine escaped involuntarily. Frequently also, in the daytime, she had attacks of giddiness, which lasted a few seconds, and during which her urine escaped involuntarily. When once epilepsy had been recognized, it was better understood how, under the influence of fits which were not noticed, her reason was sometimes seriously disturbed.

Dr. Legrand du Saule, who related the above case at a meeting of the *Société de Médecine Pratique*, mentioned also that he had seen, at Contrexéville, a young lady who pretty frequently wetted her bed, and whose tongue was wounded in some places from being probably bitten on the same occasions.

Besides the urine, the motions may be passed involuntarily, and the individual finds himself in a mess, on waking up in the morning, without having been conscious of what took place during sleep. These are circumstances which, even if occurring in persons apparently enjoying the most perfect health and unimpaired faculties, should make a medical man suspect the possibility of nocturnal attacks.

I wish now to direct your attention most particularly to other phenomena, which modern authors have allowed to pass unnoticed.

If you examine an epileptic carefully after one of his fits, or better still, several hours afterwards—the next day, for example,—you will often find on his forehead, his throat and chest, minute red spots, looking like flea-bites, which do not disappear on pressure, and have all the characters of *ecchymoses*. This is a sign of very great value, and if modern authors have laid too little stress on it, it had not escaped the notice of the ancients. "Videmus, post validos paroxysmos epilepticos [says Van Swieten] vasa cutanea minora quandocunque rumpi, et *puncta ruberrima* per totam superficiem corporis dispersa manere, quæ sensim posteà evanescunt; ubi verò rupta vasa, vel dilata eorumdem extrema, sanguinem rubrum eructaverint in tunicam cellulosam, tunc latiores maculæ et ecchymoses apparent.

Medici in praxi versati frequenter hæc symptomata observaverunt." Thus, not only are the small red punctæ I mentioned observed, but large ecchymoses also, which are produced in the same way, and apart from all contusion. This sign is, I repeat, of considerable importance, for the ecchymoses are a sure sign of an epileptic fit. Thus, an individual will tell you that on waking in the morning he felt pain and heaviness of the head, and that during the night he passed his urine or his motions involuntarily. His speech will be embarrassed, not because his tongue is paralyzed, but because it is painful and swollen from having been bitten, and sometimes cut in several places; and, lastly, you may notice ecchymoses on his forehead and throat. In such a case you can affirm that the patient has had an epileptic fit during the night.[1]

These ecchymoses give us, besides, an explanation of the apoplectiform phenomena which characterize the third stage of the fit.

I have told you already that most of the individuals seized with an epileptic fit remained for a variable period in a state of coma, and, on recovering from it, complained of headache, resembling the heaviness of the head which follows a debauch. In some cases, to which I shall revert by-and-by, the stupor is followed by nervous symptoms of another kind. They have hallucinations, become wildly delirious and maniacal—sometimes so much so, indeed, that they attempt suicide, or try to murder the persons around them. Some, again, suffer from cerebral disorders for two or three days afterwards, such as complete or partial loss of memory, incoherence of ideas, and perversion of the intellect. Now, looking at these ecchymoses of the subcutaneous cellular tissue, one may well ask whether some similar lesion of the cerebral tissue has not occurred, of the meninges, or of the spinal cord, and whether those lesions could not, in a certain measure, account for the brain symptoms which showed themselves; whether, for instance, they could not explain the paralysis which occurs in some instances, and lasts for four, six, and even ten days after a fit, disappearing then, in general, completely, until reproduced by another fit, but in some cases persisting until death.

The existence of these cerebral or spinal lesions has been ascertained in several post-mortem examinations. Calmeil, and other writers on epilepsy, have pointed them out. Not only have there been found on the surface of the brain red punctæ,

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[1 See an interesting paper, entitled "Recherches statistiques sur les Accidents produits par l'accès épileptique," by Messrs. Jules Rengade and Léon Reynaud, in *Gazette Hebdomadaire de Médecine et de Chirurgie.* Paris: Janvier, 1865.—Ed.]

like the subcutaneous ecchymoses, but blood-effusions have also been met with in the meninges, the substance of the brain or spinal cord. Softening, even, of these organs has been noticed, and an instance of this fell under my own observation, in the case of a young girl who died in the St. Bernard ward, four days after her admission into the Hôtel Dieu. She was sixteen years old, and looked of a feeble constitution. She had been for three months subject to epileptic attacks, and a near relative of hers was affected in the same way. Her fits were excessively violent, and recurred four or five times in the twenty-four hours. One of them occurred in my presence, and there could be no hesitation about the diagnosis. The convulsions lasted one minute at the most, were accompanied by contractions of the hands and feet, of the muscles of the neck, and of rigidity of the base of the chest, which rendered respiration anxious and difficult.

On the fourth day after her admission she died in a condition of profound stupor, after several attacks recurring one upon the other, and leaving rigidity of the limbs in the intervals. Dissection disclosed extreme softening of the spinal cord, the substance of which ran out through the incision made into the meninges. The spinal column had been laid open with the greatest care, so as not to injure its contents in any way, and thus avoid all source of error. On slicing the brain, a small clot, the circumference of which was beginning to soften, was found about the middle of the left posterior lobe. The brain-tissue was of normal consistency everywhere else, and slightly injected. The chief viscera presented no appreciable structural change.

Lasting apoplectiform symptoms, and paralysis which is more or less permanent, are in all probability, therefore, due in a certain measure to appreciable material lesions of the nervous centres. I hasten to add, that those lesions, congestion, hæmorrhage, or softening, cannot be regarded as causes of the epilepsy itself; nor can the serous effusions, which are sometimes met with in the cranial cavity or in the cerebral ventricles of individuals who have died after a fit, be looked upon as causes of the disease. These anatomical lesions are effects of the complaint, and no more, as it has long ago been proved by those who have studied the question. I, of course, allude to epilepsy proper, for we shall see that in cases of so-called symptomatic epilepsy the epileptiform phenomena are more or less directly dependent on the existence of brain-lesions, such as bony tumours, cancer of the brain, syphilitic or tubercular deposits, &c., which it is generally possible to diagnose during life, and which are revealed by a post-mortem examination.

With regard to idiopathic epilepsy, some authors—among

others, Bouchet and Cazauvielh—have pretended that they have always met with characteristic lesions, such as an induration of the white substance of the brain. But the cases they give by no means prove their assertion, and most, if not all, physicians are now agreed that the most delicate post-mortem investigations only give negative results respecting the organic conditions under which the disease is developed. I do not deny, however, that the cerebral disorders which constitute epilepsy depend on a material lesion of the nervous centres. When speaking of apoplectiform cerebral congestion, I gave you my opinion on that point. I then told you, and I repeat it now, that I do not conceive a functional lesion without an alteration of the organ which discharges the function; but I maintain that we have not yet been able to discover the nature of this alteration, and that the anatomical lesions which we find on dissection are the effects, not the causes, of the disease.

I shall pass rapidly over the *determining causes* of epilepsy, for the influence of the greater number of those which have been mentioned as such is far from being proved. It has thus been said that epilepsy sets in more frequently in women at puberty, about the first menstrual period, and that the cessation of menstruation is also another cause of the disease. The part played by menstruation is very doubtful, however. Epilepsy is met with at all ages, although it occurs more commonly during adolescence in both sexes. If it occurs more frequently than is generally believed in early life, as I shall show presently, it does not spare individuals advanced in years. On May 16th, 1857, Dr. Fantin (de Seineport) brought me an old farmer, seventy-three years old, who, for the last four years only, had suffered from epileptic fits. They first occurred during the night; and on waking in the morning he felt stupid, and complained of soreness of the tongue. Under the influence of belladonna, perseveringly administered for three years, the convulsive fits entirely disappeared, and he only remained subject to *fits of absence*, recurring every month, and sometimes at shorter intervals, and lasting sometimes from fifteen to twenty minutes. During these seizures he spoke incoherently; and on recovering himself he felt no fatigue, but had no recollection of what had passed since the beginning of the attack.

One of the most celebrated military men of our time became epileptic when eighty years old, and died in a fit thirteen years afterwards.

Errors in diet, excessive drink and venery, masturbation, prolonged chastity, forced intellectual labour, overstraining of the mind, violent moral emotions, &c., have often been put down as causes of epilepsy, but their real share in the production of the disease is yet to be proved. Of all these occasional causes the

influence of fright cannot be denied, and has been noted by every physician.

I have myself ascertained the fact on several occasions, but I am far from believing it to be so frequent as stated by patients and their friends. Very recently I was consulted by a Brazilian, whose first attack seemed to have been manifestly brought on by fright. Whilst on a long journey through his country he had gone to a lonely inn, where he happened to witness a quarrel between some individuals who were armed, and who, from high words, came to blows. One of the men, mortally wounded by the discharge of a gun, as well as stabbed with a knife, fell down dead in his presence. He was horribly affected by the scene, and a few days afterwards, whilst dining with a friend, he was seized with epileptic vertigo. Since that time, and for the next five years, he was every day affected in the same way. The attacks were ushered in by a sensation of great heat, beginning at the navel, and rising up the back, which was followed by absolute loss of consciousness for the space of two minutes or so. They sometimes passed away so quickly that they were not noticed by anybody near him. At the end of five years, convulsive seizures supervened, which were at first mistaken for apoplexy, and recurred at intervals of from twenty to thirty days. The vertigo disappeared from that time. He was treated by a physician at Rio Janeiro, and for the space of four years and eleven months he was free from an attack. After this interval the convulsive fits recurred again, as intense and as regular as before, persisting for six years. They then became less violent again, although more frequent, and occasionally attacked him during the night. He stated positively that no member of his family had ever been similarly affected.

It is not difficult to collect analogous instances. Thus Leuret (in his "Researches on Epilepsy," Archives générales de Médecine, 1843) states that of sixty-seven cases of epilepsy observed by himself, the first symptoms of the disease showed themselves after a fright in thirty-five.

I do not wish, however, to leave you under the impression of Leuret's too absolute doctrine. Whenever I see a case of epilepsy I carefully inquire into the cause; and although the patient in most cases imputes his complaint to fright, on closely questioning him I find, however, that in almost every instance the attacks occurred only weeks, months, and even years after the fright. I besides ascertain that this fright was not more severe or more repeated than in the case of a great many children who have never had fits. The patients only repeat what they heard from their friends, and in most cases when I can question the friends themselves and obtain the truth from them, I find that there have been members of the family affected with insanity, epilepsy, or

idiocy, and that the pretended fright only served as a pretext to hide the true cause, namely, an hereditary taint.

I do not mean either to deny the influence of emotions felt by a pregnant woman, on the fœtus in utero; but I believe that this cause has, like the rest, been extremely exaggerated. Let us now study the different forms of the disease.

I have already told you that an epileptic fit lasted rarely more than two or three minutes. I maintain this assertion, and I add further that an attack lasting from four to six minutes is of such rare occurrence that a medical man may live for years among epileptics without observing a single one. And yet you have heard of cases in which the attacks have lasted two or three days, and have terminated in death. This is the condition which has been termed *status epilepticus* at Bicêtre and the Salpêtrière. The contradiction between these facts and my proposition is merely apparent. The *status epilepticus* is characterized, not by a single attack, but by a series of attacks, and what then happens is as follows :—

The epileptic has a convulsive fit, just like a parturient woman is seized with eclampsia. In both cases the stupor which succeeds the convulsions lasts from ten minutes to three-quarters of an hour at most. But before the stupor has passed away another attack, exactly similar to the first, supervenes, and is confounded with it. Now, as the third stage of an epileptic fit is not usually regarded as distinct from the convulsive stage, the patient seems to be still in a fit, although his comatose condition is only an effect of the fit. He has not therefore got over the disturbances caused by the first attack before a second occurs, then a third, a fourth, a fifth; and in proportion to the recurrence of the fits the cerebral congestion increases, the apoplectic coma is prolonged, and extends over a period varying from two to twenty-four hours, and after a time the patient does not recover his senses at all. In some exceedingly rare cases the convulsions last a longer time than I have stated; but such cases are so exceptional that at Bicêtre and the Salpêtrière, where a considerable number of epileptics are gathered together, and where consequently from forty to fifty attacks may be seen in one day, as Dr. Calmeil did, two or three months, and even more, may elapse without a single one of the kind occurring.

In the *status epilepticus*, when the convulsive condition is almost continuous, something special takes place which requires an explanation. The patient has a fit of *haut-mal*, then every two seconds slight convulsive movements, transient and scarcely visible, affect his face, his neck, and his limbs, and these recur in the same way for the space of from two to five hours. This is assuredly a continuous convulsive attack; but it should be observed that it is no longer an attack of *haut-mal*, but quite a different and

special form of seizure, dependent on a peculiar irritable condition of the brain and spinal cord. This is what should be meant by a continuous attack; and this form, besides, occurs more frequently in cases of eclampsia than of epilepsy.

I have described epilepsy to you in its most familiar form, and it now remains for me to say that the *haut-mal* varies in intensity, in violence, and in suddenness of seizure. Some individuals are struck down without any premonitory symptom, and without uttering a cry. In others, whilst they are being spoken to, their knees gradually bend, and they fall down senseless, without the least convulsive movement. Although rare, such cases are yet met with.

Some time ago, a child affected with this singular form of epilepsy was brought to me. His friends were telling me how he was attacked four, five, and even six times in an hour, when he suddenly slipped from the armchair in which he was sitting, and fell down on the carpet. I examined him carefully, but detected nothing approaching to convulsion.

Another individual, about whom I was also consulted, had similar attacks two or three times a week. The seizures at first set in with hallucinations which lasted half a minute, during which time he stared vacantly, with his arms hanging down by his side. The symptoms then became modified, and he lost his senses during attacks which lasted about ten minutes. The case being mistaken for one of cerebral congestion, leeches were applied, but after this a second attack came on, which was accompanied by convulsions of the face and rolling of the eyeballs.

This form of epilepsy consists, then, in mere giddiness, and seems to leave behind it scarcely any consequences, any immediate ones at least. The patient, on getting up, looks a little bewildered, but is soon able to resume the interrupted conversation, as if nothing had occurred. The attack does not proceed beyond the first stage, and although strong enough to prostrate the patient, it does not pass on to convulsions.

In other cases, on the contrary, the first stage is absent. The epileptic falls down, his upper limbs, sometimes his eyes alone, are agitated convulsively, and he then gets up almost immediately scarcely feeling a little stupid, and somewhat mentally confused for a short time.

In other instances, again, the fit occurs as usual, but is extremely slight. There are tetanic convulsions, but only for an inappreciable time; clonic convulsions follow, and after a few seconds the stage of stupor comes on, and is as transient and as slightly marked as the preceding. The patient then gets up, and the attack has scarcely lasted a minute.

These are very different forms from those which we were

studying just now; and they are, as it were, transitions between attacks of *haut-mal* and other manifestations of epilepsy to which I am now about to call your attention more particularly. Keep these facts well in mind. No case of epilepsy is more genuine than that in which the fit occurs quietly, without any extensive movements, and without much noise. If an attack of *haut-mal* can be sometimes so well feigned as to deceive those who are not thoroughly familiar with it, it is quite different with the small seizures, with the fits of vertigo, which I am now proceeding to consider.

§ 2.—Epileptic Vertigo.—Aura Epileptica.—Partial Epilepsy.—Angina pectoris.—Painful spasm of the face.

Vertigo, gentlemen, is a manifestation of epilepsy which is least familiar to medical practitioners, and errors of diagnosis are committed every day, which may be followed by dangerous consequences, through a very grave disease being represented as a trifling ailment.

Let me first cite a certain number of instances of vertigo, and thus attempt to show you the numerous forms which it may assume. But remember that however various the forms, the disease is always the same, and that these transient, strange phenomena which sometimes consist only in giddiness, in a sort of astonishment, in ecstasy, or in what has been termed *a fit of absence*, are identical in their nature with the violent convulsions which characterize an attack of *haut-mal*. Nay more, vertigo is to a certain extent much more characteristic of epilepsy than convulsions are. The latter, indeed, may be a symptom of other diseases which, however much they differ from epilepsy, are frequently confounded with it. Thus, in females, hysterical fits resemble epilepsy so closely as to be mistaken for it, and those who have had occasion to observe a good many cases of hysteria, as at the Salpêtrière, know how difficult it is in some cases to distinguish between the two affections. Epileptic vertigo, on the contrary, as well as the vertigo of eclampsia, has a special physiognomy, which, when once studied and looked out for, cannot be confounded with anything else.

Inquire carefully into the case of an individual suffering from this form of epilepsy, and especially if the patient be a youth or a child, you will recognize a more or less distinct manifestation of the disease by the symptoms he will describe to you.

I have already pointed out the transformation of the symptoms into one another. In general, vertigo precedes the convulsive form, but the reverse sometimes obtains. The *haut-mal*, which had been the first manifestation of the disease, becomes modified; the attacks diminish in violence, and the individual becomse subject to *petit-mal* only (another name given to epileptic vertigo)

An instance of this, as you know, occurred in the case of a young man lying in bed No. 18, St. Agnes ward. Nor is it uncommon to see convulsive attacks and vertigo develop themselves simultaneously, or the latter appear at least in the intervals between the former, or even usher them in.

A gentleman came one day, from Berry, to consult me. During the short time he remained in my consulting-room, he was seized with vertigo, characterized by jerking bursts of laughter. The fit lasted a few seconds only, and he immediately recovered himself; but he seemed very much surprised when I asked him why he had laughed: he was not conscious of what he had just been doing. The convulsive attacks to which he was subject were almost always ushered in by these vertiginous seizures.

The concomitant existence, or alternating production, of these various morbid phenomena, clearly point out their connection and their identical nature.

Let us now rapidly review some of the forms assumed by epileptic vertigo, keeping in mind that these forms vary indefinitely, and that it would be vain to try and describe them all.

You remember a young girl, aged 16, who, for a long time, was in St. Bernard ward, and to whose case I have already alluded in a former lecture. You remember the seizures to which she was several times subject in the course of the twenty-four hours, and which I witnessed on several occasions with you, when going round the wards. She suddenly lost all consciousness of her acts, and dropped, or more frequently threw away at a distance, anything she might be holding. Sometimes she would then jump about, turning round her bed as if she were looking for something; at other times, she would fall down, whilst her face grew pale for a moment, and her eyes rolled convulsively upwards under the upper eyelid, and looked strangely fixed; on other occasions, again, she would keep clapping her hands rapidly. If she happened to be seized in bed, she sat up, and took hold of the bedclothes, as if she wanted to cover herself up. The attack scarcely lasted half a minute, and as it passed off, she called out "It is over." Very slight and very transient stupor then followed. But a very remarkable circumstance in this case was that if an attempt were made to take from her an object which she might be holding at the time of her seizure, she rushed on in a kind of rage, in order to gain possession of it, and struggled until the fit was over.

She stated that her illness dated from the previous year only, and had set in with vertigo, or what she termed "fits of surprise." She had as many as a hundred attacks in one day, and occasionally had convulsive fits. She had no warning whatever. Her father and mother had never suffered from any analogous complaint, but a sister, now dead, had been epileptic.

Thus, in the majority of instances, suddenly, and without any premonitory symptom, as in an attack of *haut-mal*, the individual subject to epileptic vertigo feels a kind of astonishment, becomes absent, as it were. If he is engaged in conversation at the time, he suddenly stops in the middle of a phrase, and with eyes fixed, looking bewildered, he neither sees, hears, nor feels anything. He is in a kind of ecstasy, and yet he does not fall down. If he has an object in his hands, he drops, or convulsively throws it away from him. The whole lasts from two to four seconds, and sometimes more; the attack is then over, the patient recovers himself completely, resumes his occupation or the conversation in which he was engaged, and has no suspicion of what has occurred.

Dr. Taupin once asked me to meet him in consultation about a little girl, six years old, who had been ill for five weeks, and whom I had already seen. He told me that he had himself witnessed two attacks which had occurred at dinner-time, and the girl's mother also gave an excellent account of what happened. The child, whilst at play or at dinner, stopped suddenly, and turned her head slowly to the right, with her eyes open and fixed. There were no appreciable convulsions, and no distortion of the face. Sensation was so completely abolished that her skin could be pinched or pricked with a needle without her seeming to feel pain. She remained in that condition for the space of four or five seconds, and then recovering herself looked somewhat bewildered and cross. Generally also she then expressed a wish to move about, requesting her mother to take her into the next room. But in a few seconds she was perfectly herself again; and, after drawing a deep sigh, she returned to her play, or went on eating, as the case might be. The attack of vertigo may last a longer time, however, and may consist in, or be accompanied by, more or less marked delirium, manifesting itself by words and acts.

On another occasion, I was consulted about a little girl, aged four, who, for the preceding fortnight, had presented symptoms like the above on every other day. She was otherwise in excellent health, had a precocious intelligence, and related very well what she felt. She experienced something like a general shock, according to her own account, and then became unconscious. Her mother, however, told me that her face then assumed a singular expression of cheerfulness and vivacity in some cases, whilst in others the child looked stupid. After scarcely a minute had elapsed, she exclaimed that she was frightened, acted in a strange and disorderly manner, and spoke incoherently. These hallucinations were sometimes prolonged for seven, eight, and ten hours. Within two days the attacks had recurred twice in the twenty-four hours. The

mother added that she thought her child's intelligence was getting impaired.

A medical man, practising at Versailles, sent a young girl to consult me, in December, 1860, whose mother and grandmother were healthy, but whose aunt and great-aunt, on her mother's side, were subject to epilepsy. She herself suffered from attacks of vertigo, which were so frequent that I saw four or five of them whilst she was in my consulting-room. She uttered a plaintive cry, aud suddenly placing her hand on the pit of her stomach, she slowly turned her head over to one side. Her eyes were at the same time fixed, her face was slightly distorted. Before a minute had elapsed all seemed to be over, and she then got up, looking bewildered, staggered, and sometimes fell down. If any one came near her, she seemed to feel a sort of terror. I questioned her quickly; but she opened her mouth and made signs that she could not speak; I asked her to put her tongue out, and to move it about, but she was unable to do so. A few moments afterwards she uttered a few inarticulate words, and on my insisting to make her speak, her speech became gradually less embarrassed, and then perfectly natural. The attack lasted four or five minutes altogether. She was very intelligent, and described her sensations very well. She stated that on the accession of the fit she felt acute pain in the epigastrum, which almost instantly extended to the tongue, when it became very intense. She then lost her senses for one or two minutes, and, on beginning to come round, she was prevented from speaking by a kind of painful paralysis of the tongue, which gradually passed off.

Again, an individual who is subject to epileptic vertigo may, whilst playing at cards, and holding in his hand a card which he is going to throw down, suddenly become motionless, shut his eyes or stare before him, and then, after drawing a deep sigh, he may continue to play. These, gentlemen, are types of epileptic vertigo, and I might multiply instances of the same kind. But there are other and different forms, which I will now point out to you.

In the above cases, the patient is isolated from the external world; he sees, hears, and feels nothing, and remains perfectly motionless, in a kind of ecstasy. In some instances movements resembling those of mastication are performed, followed by the same guttural sound as when saliva alone is swallowed. In other instances, there is some mental confusion or disorder which lasts a few seconds, a few minutes even, but which escapes the notice of by-standers. Lastly, there are cases in which the epileptic may complete the movements he has begun, and even perform new ones with a certain degree of regularity, although he is perfectly unconscious of his acts.

I have on several occasions cited the case of a priest who, whilst officiating as deacon, and incensing the bishop from the thurible, was seized with epilepsy, and still continued swinging the censer, although his head was so strangely twisted round, and his face so contorted, that the fit attracted everybody's attention. He was subject to vertigo, and had been often attacked in the pulpit, or at the altar, whilst officiating. The attacks, however, were so transient, that he had never been obliged to interrupt his sermon or go away from the altar. But as during the fit he sang in a strange manner, and had on some occasions uttered incoherent words, these acts being considered undignified in a priest, he was of necessity suspended. He came to consult me, and told me himself the above details.

I have already mentioned to you, in a preceding lecture, the case of a young amateur musician subject to epileptic vertigo, and who has sometimes a fit whilst playing the violin. Strange to say, he goes on playing during the attack, and although he is perfectly unconscious of everything around him, and neither hears nor sees those he is accompanying, he still plays in time. It would seem as if his will were powerful enough to direct the movements of his hands for a given, though very short time, and as if those movements were guided by memory, the patient performing without a fault the musical phrase which he had read just as his mind became affected.

Many of you may recollect having heard me relate the following case :—An architect who resides in Paris and has long been subject to epilepsy, does not fear to go up the highest scaffoldings, and yet he is perfectly aware that he has often had fits whilst walking across narrow planks, at a pretty considerable height. He has never met with an accident, although when in a fit he runs rapidly over the scaffoldings, uttering, or rather shrieking out his own name in a loud, abrupt voice. A quarter of a minute afterwards he resumes his occupation, and gives his orders to the workmen; but unless he be told of it, he has no idea of the singular act which he has been committing.

I once knew a gentleman of superior intelligence, the president of a provincial tribunal, who was subject to epileptiform symptoms, but had never had an attack of *haut-mal*. Some of his relations were of unsound mind, his sister among others. One day, whilst the court was still sitting, he got up, muttering a few unintelligible words, went to the council-room, and returned a few seconds afterwards, unconscious of what he had done. When his colleagues asked him where he had been to, he did not recollect having moved from his place. Shortly afterwards, as he was getting up in the same manner, the usher was told to follow him. He was then seen to enter the council-room, and make water in a corner, after which he returned to the court,

perfectly ignorant of his incongruous act. He noticed himself, however, that for a few minutes after these attacks his mental faculties were somewhat impaired. I heard of these facts from himself and from his father-in-law. I did not conceal from the latter of what grave import they were, and I recommended that the patient should resign his post. He had some difficulty in deciding upon this step, but one day whilst in court he got up, walked about, and spoke incoherently to the people around. Almost immediately afterwards he resumed his seat, and without any appreciable mental disturbance, continued to lead the debates. His conduct, however, had caused such surprise that his colleagues told him of it, and fearing lest his fits of absence should be used as reasons for quashing his judgments, he sent in his resignation.

It is this same gentleman who, as I told you in a former lecture, suddenly left a meeting at which he was discussing some historical questions, at the Hôtel de Ville, ran out into the open square outside, without his coat and hat, avoiding carriages and the passers-by, and on recovering himself returned to the meeting. In a certain measure, his condition was somewhat analogous to somnambulism. Sometimes, when engaged in reading, he would suddenly cease, and would repeat with volubility the last verse or the last portion of the phrase at which he had stopped. His physiognomy wore an unusual expression at such times, but he almost immediately took up his book again and resumed his reading.

You will not only meet with persons who are able to perform certain acts during the attacks of epileptic vertigo, but also with some who can answer when spoken to, although they are not conscious of their answers. Their condition may be compared to somnambulism, or, better still, to what happens in the case of certain individuals who answer questions during sleep, but do not recollect anything when they wake up.

I attended some time ago a young lady suffering from this vertiginous form of epilepsy. During the attacks, her face sometimes wore an expression of terror, sometimes of anger. She made no answer when spoken to quietly, but if addressed abruptly and in a commanding tone, she answered curtly and in a loud voice. She then suddenly paused, and, if addressed in the same way again, she looked bewildered for a while. Each attack lasted from fifteen to thirty seconds, and when it was over she had no recollection whatever either of what she had been asked, or of the answers she had made.

I knew a child who used to exclaim, "Go away, go away," whenever it was attempted to make him, during a fit, inhale some ether or ammonia, the smell of which he disliked.

I shall next draw your attention to other disorders of innerva-

tion belonging to the same group as those we have just studied—I mean what has been termed *aura epileptica*.

These singular disturbances of the nervous system, which sometimes usher in epileptic seizures, are perhaps more frequent in cases of *grand-mal* than of *petit-mal*. In some instances, however, which belong, therefore, to the vertiginous and not to the convulsive form, they alone constitute the attack. A peculiar sensation—which the individual compares to a kind of wind or of vapour, or to tingling—starts from some portion of his body, spreads upwards, and, on it reaching his head, he suddenly falls down in a fit.

When the *aura* begins in the hand or in the arm, the patient feels the strange sensation running along the length of the limb, which is sometimes convulsively agitated to a scarcely appreciable degree. It rapidly spreads higher up, affects the head, and the fit then begins. You will observe this phenomenon in a large number of cases.[1] More or less transient in character, it lasts from one second only to a minute sometimes. In some cases, it does not merely consist in a strange sensation, but in an acute pain, affecting the hand or the foot, running the same course upwards in both cases, and followed by the fit when the head is reached.

In other instances the *aura* is attended with appreciable material changes in the part from which it first started. A local determination of blood may occur in the finger, for instance, causing it to swell, reddening the skin, and rendering it successively, within a very short time, red, and of a more or less deep violet colour; or again, the skin may become excessively pale after having been injected for some time. The swelling is real, not apparent; for rings, previously easy, suddenly become too tight for the fingers.

The *aura epileptica* may be again characterized by sudden convulsive phenomena, as in the case of a little boy, who was in my ward at the Children's Hospital, in 1848. On several occasions

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[1 Dr. Sieveking states that out of fifty-eight cases of epilepsy of which he had preserved careful notes, thirty showed some indication of the approaching paroxysm.—("On Epilepsy and Epileptiform Seizures; their causes, pathology, and treatment." London, 1858.) Romberg (*op. cit.*, p. 197) says that he found premonitory symptoms occur in about one half of his epileptic patients.

See "Physiology and Pathology of the Central Nervous System," by C. E. Brown-Séquard, M.D., sect. xi. p. 178, *et seq.*, on the importance of ascertaining the presence of an aura, and on the existence of *unfelt auras*. These latter may be detected by "the application of a very powerful galvanic current, with dry conductors on the various parts of the skin, when the patient expects to have a fit. A fit is produced by the galvanization of certain parts of the skin. In the case of the extremities, another and better means consists in applying ligatures on each limb alternately, just before a fit is expected. If the fit does not come on, it is very probable that it depends on the irritation of an unfelt aura."—ED.]

he was seized whilst I was going round the ward, and I heard him call out, "I am taken with it." His hands were first moved involuntarily, the muscles of his face were next affected, and convulsions followed. The case terminated fatally, and, on making a post-mortem examination, I found tubercles in the brain, which were the cause of the epileptiform seizures I had observed during life.

A year ago you had occasion to see a similar case—that of a young man lying in bed No. 9, St. Agnes ward, whose epileptiform attacks were doubtless owing to a cerebral tumour. He remained in my ward for a month, and during that period I saw him in eight or ten fits. They were ushered in by pain suddenly attacking the foot, which, on being exposed, was seen to be arched and agitated convulsively. The convulsions then extended to the leg; and on his calling out next, "My arm is affected," I could see the arm jerked spasmodically. The convulsions lasted from fifteen to twenty seconds, during which his intellect was perfectly clear, and he continued to talk quite rationally. The *aura* gradually, but very quickly, extended to the head, and the poor fellow then became unconscious. In both these cases the epilepsy was symptomatic; but, as I shall show you hereafter, genuine and symptomatic epilepsy bear the greatest resemblance to each other—I may even say a complete resemblance with regard to the manifestations which constitute the seizures.[1]

The *aura* may be *visceral*,—that is to say, it may start from some internal organ. It is often misunderstood in such cases, and gives rise to errors of diagnosis, of which you should be told, in order to avoid them. A young person, at the onset of a

[1] Romberg gives the name of "*motor aura*" to the convulsive phenomena which sometimes precede a regular epileptic fit. He states that "the motor aura is not unusual; it occurs as twitching or contraction of the fingers or toes, or of one hand, one foot, one leg, or as spasm of the sterno-cleido-mastoid, drawing the head down on one side. In some patients I have seen twitchings of the muscles of the alæ nasi and the upper lip, which are supplied by the facial nerve. In one, there was the extremely rare phenomenon of spasm of the auricular muscles."—("A Manual of the Nervous Diseases of Man," by Moritz Heinrich Romberg, M.D., translated and edited by Edward H. Sieveking, M.D. vol. ii. p. 198.)

Dr. Russell Reynolds ("On Epilepsy," p. 96) details an interesting instance of *motor aura*. "The patient had jerking of the left leg, which was drawn up behind him. Elongation of the muscles arrested the fit."

An unmarried woman, aged 30, who is at present attending the National Hospital for Paralysis and Epilepsy, as an out-patient, has been for years subject to fits, which are always preceded by spasmodic closure of the left hand. If the fingers be forcibly extended, and the hand kept open, the fit is warded off.

In another case under my care, that of a little girl, aged 12, Annie S. . ., the fits are constantly preceded by pain in the little finger of the left hand. This creeps up the arm, and is attended with convulsive agitation of the limb. Firm pressure round the upper part of the arm prevents the fit.—Ed.]

fit of *haut-mal*, used to feel an acute pain in the heart, soon followed by violent palpitations, then by giddiness, and by a tendency to syncope.

Seven or eight years ago, I was consulted for a child about ten years old, who, four or five times a day, before as well as after a meal, always without any appreciable cause, complained suddenly of a sensation of pressure in the pit of the stomach, soon followed by vomiting. Immediately upon this he felt violently giddy, and turned deadly pale. These phenomena lasted altogether for about a minute. The medical man who had sent the patient to me, believing him to be suffering from dyspepsia, had vainly tried every means for combating it. The suddenness of the attack, the violence of the pain, which the child described perfectly, the accompanying sense of suffocation, the momentary impairment of the intellect, the pallor of the integuments, and, lastly, the rapidity with which these phenomena disappeared, made me write to the usual medical attendant that the case was certainly one of epilepsy. I therefore advised him to keep strict watch over the boy, adding that I was convinced that sooner or later this neurosis would assume more distinct characters, which would clearly point to its real nature. The boy's father refused to believe in my diagnosis, and his medical man concurred with him. The following year, however, I was again consulted; but this time my fears had been realized, and my diagnosis confirmed, by repeated attacks of epilepsy, from which the boy had suffered.

This *visceral aura* escapes the observation of the physician all the more easily from its simulating other affections in a numerous class of cases. If it begin in the stomach or the uterus, or if it be accompanied by that sense of constriction in the throat which is assigned as one of the characteristic symptoms of hysteria, especially if occurring in a young female, it may be confounded with the *aura hysterica*. Careful observation, however, and a rigorous analysis of the symptoms, will enable the physician to distinguish the one affection from the other. Although the *aura hysterica* seems to start from the same point, from the same organ, as the *aura epileptica*, it does not spread with the same rapidity, nor does it set in with the same suddenness. Hysterical spasms, for instance, persist a longer time than the epileptic sensations. These latter, whether consisting in giddiness or convulsions, scarcely last a few seconds—one or two minutes at the most,—although they leave behind them the apoplectic stupor I have already mentioned. In hysteria the duration of the symptoms is entirely different, and when they have passed off the patient feels nothing which can be compared with the bewilderment of an epileptic.

In general, the sensations which constitute the *aura epileptica*

spread *from below upwards*; that is to say, they begin either at the extremity of a limb, or in some point of the trunk, and go up to the head. In some cases, however, the *aura* runs a different course, from *above downwards*. It begins in the head, in the shape of giddiness or of pain, and, descending with rapidity, spreads to the limbs.[1]

In some rare instances, the aura may be both ascending and descending at the same time.

Ch. Bonet ("Sepulcretum Anatom." lib. i. sect. xii. p. 291) mentions the case of a man, aged 50, whose left inguinal region first swelled, and who next felt a sort of creeping sensation descending gradually along the thigh and affecting the foot; once there it ascended with extreme rapidity to the head. These singular phenomena have been long ago pointed out by observers. Morgagni, in the third letter of his work, "De Sedibus et Causis Morborum," cites several cases observed by himself, or reported by contemporary or former authors, and has a long dissertation on the subject. He quotes, among others, a case of Tulpius, in which a fit was brought on by pressing with one finger the region of the spleen.[2]

I have told you that the *aura epileptica* is sometimes the only manifestation of epilepsy. Indeed, it sometimes happens that it is entirely limited to the point where it first shows itself, or at least does not spread far. It does not spread to the brain, and causes none of the phenomena which more essentially characterize the disease. Those are cases of what might be termed *partial epilepsy*. When I was physician to the Necker Hospital, I had under my care a woman, who suffered from these attacks of convulsive aura, four, five, and even seven times in an hour. The aura began in her leg, and was limited to one-half of the body; the convulsions were violent, painful, and affected the trunk, the arm, and the face. Whilst they lasted, she cried out with the awful pain she felt. Her mind remained perfectly clear, although her speech was somewhat embarrassed, owing to the convulsion of the muscles of her face, and, probably, also those of the tongue. The attack lasted from a minute to a minute and a half; after which time she recovered completely. She was rapidly cured by belladonna.

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[1] In the case of a young girl, aged 18, whom I had lately under my care, the aura began in the right wrist, and extended quickly to the tips of the fingers, producing there a sensation as if the nails were pulled off at the roots, upon which the patient became unconscious and fell into a convulsive fit.—Ed.]

[2] The following is an instance of a very unusual form of aura in a youth 16 or 17 years old, under the care of Dr. Radcliffe, at the National Hospital for Paralysis and Epilepsy. The fits were always preceded by a painful sensation referred to the base of the tongue, and exactly localized in the *foramen cæcum* of Morgagni, at the apex of the V formed by the *papillæ vallatæ*.—Ed.]

A good many cases of *Angina pectoris* are certainly a form only of partial epilepsy, as I shall prove to you hereafter, when treating of that disease. I shall show you, that if the awful pain which characterizes this affection generally starts from the præcordial region, and from there shoots through the chest to the throat and to both arms, mostly the left arm, causing numbness of the limb in which it has been most intense, and attended with a feeling of anxiety and undescribable terror, the pain may, in certain instances, follow a contrary course—may, for instance, begin in the arm, and subsequently radiate to the throat, attack the præcordial region, and bring on the sense of anxiety.

The young man, lying in bed 18, St. Agnes ward, presents us with another instance of partial epilepsy; and in his case, the order in which the phenomena occur can escape nobody. His complaint, as you remember, set in at first with convulsive attacks, which gradually became less and less violent, and at present they consist in convulsions of the face, exclusively confined to the left side, and unattended with loss of consciousness. He feels at the top of the chest a painful sensation, which suddenly extends from the trunk to the face, producing a quivering of the latter. In this case there is also embarrassment of speech, due to the involuntary contraction of the muscles of the tongue and cheeks.

Perhaps we ought to place by the side of these partial epilepsies an affection the study of which is highly interesting, and which I mean to bring before you at a future period. It is that affection which I have named *epileptiform neuralgia*, between which and the different forms of *aura*, and, consequently, the other forms of epilepsy which I have pointed out to you, a connexion may in some measure be traced.

I have thus spoken at great length, gentlemen, of epileptic vertigo, of the various kinds of aura, and of partial epilepsy, because it seemed to me of the highest importance that your attention should be drawn to them; more particularly as, generally speaking, the vertiginous form of epilepsy is the one more frequently observed.

Another characteristic of this form of the disease is the great frequency of the fits. The patient may have as many as 50 and 100 attacks in the course of the 24 hours, whilst this is never the case with the convulsive form. Besides, epilepsy presents the greatest irregularity in its course and its progress, in the frequency of the seizures, not only in different individuals, but also in the same person.

I shall not revert to what I have already told you concerning the exclusive preponderance of the convulsive attacks in some individuals, and of vertigo in others, or of their respective transformation, or again, their simultaneous existence. You recollect my telling you also of patients being attacked in the day-time

only, or alternately at night and in the day-time; whilst others, in much more numerous instances than is generally believed, are only seized at night.

With regard to the frequency of seizure: some persons may, in the whole course of their lives, have very few attacks, these attacks recurring at variable intervals; or they may have a single attack only. Sometimes the fits come on periodically, at nearly equidistant intervals, or they follow one another in rapid succession, as in a series, and then cease for a pretty long time. In other instances, they recur every two months, every month, every fortnight, every week, and even every day. They may again be so frequent, as in the condition termed *status epilepticus*, that they run into one another as it were, and simulate a continued attack which lasts over two or three days.

The fits of *petit mal* being so considerably more frequent than those of *grand mal*, it is perfectly conceivable then, that dementia should be more rapidly brought on in such cases, since the central disorders which precede, follow or accompany the epileptic seizures, being repeated at shorter intervals, more quickly produce impairment of the intellectual faculties, as an almost fatal consequence.

§ 3.—On the relations of Epilepsy to Insanity.

"Epilepsy," says Esquirol, "is a dreadful complaint, not only on account of the violence of its symptoms (in the convulsive form), and not only driving one to despair on account of its incurability, but also because of its fatal influence on the physical and moral condition of its victims. The functions of organic life are impaired and become languishing. Epileptics are subject to cardialgia, flatulence, spontaneous lassitude, and trembling; they take little exercise, and become either obese or emaciated; they have a tendency to venery and onanism. Perhaps the excesses they commit are the cause of the organic lesions and of the disorders which manifest themselves when epilepsy has lasted a long time. They do not, as a rule, live to an advanced age. The cerebral functions, the intellectual faculties become more and more degraded.[1]"

You are well aware that this fatal influence of epilepsy on the intellectual faculties, of which dementia, idiocy, and general paralysis are the ultimate expression, is a well-known fact, which has been long ago pointed out by observers.

If there have been epileptics, who, in spite of more or less frequent attacks, have retained, to the end of even a pretty long career, not only the fulness of their reason, but also the full force of their intellect, and like those men of genius, whose names his-

[1] Esquirol, "On Mental Diseases," vol. i. art. Epilepsy, pp. 282, 283.

tory has handed down to us, have preserved that superior intelligence which enabled them to rise above the ordinary level of their fellow-men, instances of this kind are too exceptional to invalidate in the least the general law.[1] In the great majority of cases, although at the beginning, and when the attacks are infrequent, the patients are in full possession of all their faculties, although "a marvellous aptitude for conceiving things quickly, or viewing them under their most brilliant and poetical aspects, may distinguish some of them," as Dr. Morel[2] has remarked, yet in proportion as the fits recur and increase in frequency, in proportion as the disease progresses, the faculties fail, are impaired, become gradually extinct, and insanity follows.

Often, also, in individuals whose intellectual activity is perfect, a singular changeableness of feeling, of temper, and of character, violent fits of passion which they cannot master, point to a particular mental condition, which, in the greater number of cases, will be followed by physical phenomena of a more distinct character, but always of the same order, as well as by more serious cerebral disorders, such as attacks of delirium, sometimes transient, sometimes prolonged, and then specially deserving the name of *epileptic insanity*.

In general the cerebral disturbance is connected with the so-called physical symptoms of the disease, namely, the attacks of convulsions or vertigo, and manifests itself in the interval between the seizures, at their onset, or, more commonly, more or less immediately after them. In some cases, however, these psychical phenomena seem to be the only manifestations of epilepsy. On

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[1 It is well known that three very remarkable men—Cæsar, Mahomet, and Napoleon—are said to have suffered from epilepsy. With regard to the first of these, the Emperor Napoleon III., in his recently published "Life of Cæsar," rejects the idea that the "two nervous attacks from which the illustrious Roman suffered, one at Cordova and the other at Thapsus, were attacks of real and genuine epilepsy."—("Vie de César," p. 258.)

Napoleon I. is said to have suffered something like epilepsy during sexual intercourse. The circumstance is mentioned by Dr. Watson ("Lectures on Medicine," vol. ii. p. 641), who adds that this is not very uncommon in persons subject to that disease.

Romberg (*op. cit.* p. 213) also observes, that there are individuals, especially of the male sex, who are seized with epilepsy after every coition.

Esquirol ("Maladies Mentales," p. 300) cites cases also in which the fit always occurred after connection.

Dr. Russell Reynolds ("On Epilepsy," p. 92) reports the case of a young man, æt. 25, in whom epilepsy was brought on apparently by sexual excess, and who had an attack during intercourse.

In a case which came under my own observation, that of a lad aged 18, subject to epilepsy probably due to Onanism which he practised every day, and sometimes two and three times a day, a violent epileptic fit occurred on one occasion whilst he was masturbating.—Ed.]

[2] B. A. Morel (de Saint Yon), "A Treatise on Mental Diseases." Paris, 1860, p. 696.

the whole, the course which they run is very characteristic, and possesses considerable medico-legal importance.

This point in the history of epilepsy has, within the last few years, been the subject of special study, and has given rise to numerous memoirs, among which I shall mention that of Dr. Jules Falret.[1]

"The intellectual disorders observed in epileptics," says the author (from whom I borrow the greater part of what I am now going to tell you), "may be divided into three principal categories: 1st, those which, manifesting themselves in the intervals between the attacks, are independent of these, and constitute the habitual mental state of epileptics; 2nd, those which occur temporarily before, during, or after the attack, and may be considered as epiphenomena of the attack itself; 3rd and last, intellectual disorders, more or less prolonged, which coming on in paroxysms, either directly connected with the convulsive or vertiginous phenomena, or occurring independently of these, specially deserve the name of epileptic insanity."

Although some epileptics may, through life, be in full possession of all their faculties, and may manifest in their conduct no sensible change, at least in the beginning, or when they are subject to infrequent attacks only; in the vast majority of instances, however, those, particularly, who are subject to more or less repeated attacks, present in *the interval between the seizures*, certain phenomena manifestly dependent on a *particular mental condition*, which cannot yet be termed *insanity*.

The predominating element in these phenomena is an extreme changeableness of temper and of mental dispositions; a true intermittence of the psychical phenomena referable to the affections and the temper, or belonging to the intellectual faculties.

Thus they sometimes look sad, peevish, desponding, as if under the influence of grief or of shame, arising from their awful complaint; at other times, on the contrary, they have inward sensations of ease and satisfaction which prompt them to harbour thoughts of rash undertakings, or to conceive projects which they can least realize in their sad condition. Sometimes they are querulous, inclined to controversies, to discussions, to quarrels, and even to acts of violence; at other times, on the contrary, they evince a gentle, benevolent, and affectionate disposition, and religious sentiments of submission and humility as exaggerated as their previous behaviour had been.

"The same contrasts which are observed in their feelings, are also noticed in the degree of their intelligence, and in the nature of the ideas which occupy their minds. Nothing is more mobile

[1] Jules Falret, "De l'état mental des Epileptiques" ("Archives Générales de Médecine," Décembre 1860, Avril et Octobre 1861).

than their mental dispositions and the level of their intelligence: they sometimes suffer from mental confusion, failure of memory difficulty of attention and comprehension. They have great difficulty in collecting their thoughts, and are themselves conscious of the obtuseness of their intellect and the confusion of their ideas. At other times, on the contrary, they evince real intellectual activity, a rapid circulation of ideas, which corresponds with a certain degree of cerebral excitement. They can, at such times, devote themselves to uninterrupted study, of which they are incapable at other times, and remember certain facts and certain ideas which, on other occasions, they seemed to have completely forgotten."

"This irregularity in the state of their feelings and the degree of their intelligence is necessarily reflected in their talk and in their acts. Hence the excessive variability of their behaviour towards those about them. For a certain period of their lives they are laborious, punctual, attentive to the duties of their profession, obedient and docile, and those who live with them or who employ them find their intercourse agreeable, or are pleased with their services. But at other times, their conduct becomes suddenly modified, and presents the greatest irregularities. They are then incapable of fulfilling the duties confided to them, become negligent, lazy, and indolent. They forget the most elementary things, waste their time, or wander here and there, without aim or object in view; and are themselves conscious of the vagueness and confusion of their ideas. The most deplorable tendencies and the worse inclinations develope themselves in them at the same time: they become liars and thieves; they pick up quarrels with those around them, complain of everything and of everybody; are very easily irritated for the slightest cause, and even frequently commit sudden acts of violence, which, in most cases, have not the excuse of provocation on the part of the victims to those acts."[1]

We have seen, gentlemen, that in the vast majority of cases, if not in all, epileptics are completely unconscious *during their seizures*, and that this loss of consciousness is even one of the characteristics of the malady. We have also seen that in some cases, instances of which I related to you, the patients, although uncognizant of the outer world, utter certain words and perform certain actions as what obtains in natural somnambulism. I will add that, whilst some individuals have no recollection whatever of what has occurred, others remember more or less vaguely the ideas which occupied their mind, and have a confused notion that they were then, as it were, "under the influence of a painful dream, of intense pain, or of deep remorse; or, again, of a sense of some unavoidable misfortune, which they could not account for."

[1] Jules Falret, *loco citato*. Dec. 1860, p. 669, *et seq.*

These singular intellectual disturbances principally occur in those epileptic attacks which, according to J. Falret, hold a medium place between simple vertigo and convulsive fits, and which are incomplete with respect to the disorders of movement as well as the loss of consciousness. But the psychical phenomena which may show themselves *before* or *after* the fits are much more interesting to study, and much more important to know. By the side of individuals who are seized suddenly, without any premonitory symptoms, you will observe others in whom appreciable changes of temper foretell, like clouds, forerunners of a storm, that a fit will occur more or less shortly. "Thus, for example, certain epileptics become sad, peevish, quarrelsome, irritable, often for several hours before a fit; others complain of slowness of conception, of failure of memory, of obtuseness of ideas, of a kind of hebetude, or physical and moral prostration, which to those used to their society or to themselves are sure signs of an approaching fit. Others, on the contrary, are unusually gay, have an exaggerated sense of physical and moral well-being, an excessive confidence in their own strength, and sometimes even get into a state of loquacious restlessness which may be pushed on to maniacal excitement or to violent bursts of passion.[1]

"Apart from these premonitory symptoms, which may come on at a variable time previous to an epileptic seizure, there are other prodromata of the same order, a sort of *intellectual aura* which precedes the convulsion by a few minutes only, and constitutes its first symptom in a certain measure." These prodromata consist in hallucinations, illusive sensations, varying indefinitely in different individuals, but recurring in the same person with singular uniformity. Thus, a young person, subject to epilepsy, told me that at the beginning of a fit she heard voices and sounds which were remarkably harmonious and melodious.

Other patients declare that they hear sounds of bells, or a voice uttering the same word in a determined tone. Others, again, always smell a particular smell, or see a ghost, flames, fiery circles, frequently red or purple objects, or (as in the case of the Brazilian whose history I related to you) the objects around them look unusually bright and beautiful, and form a magic spectacle. These strange and excessively variable sensations resemble those of individuals under the influence of Haschisch. Lastly, in other cases, the intellectual aura consists in the recollection of a fact, or the reproduction of an idea, which on a former occasion either caused, or at least accompanied the

[1 See Dr. Radcliffe's graphic description of the "Premonitory Symptoms of Epilepsy" in his work on "Epileptic and other Convulsive Affections of the Nervous System."—Ed.]

fit. "Many persons," says Dr. J. Falret, "who have become epileptics after strong moral emotions or intense terror, see again in spirit, or before their eyes, on each succeeding seizure, the painful circumstances or the dreadful scene which first produced their complaint."

A young man, aged seventeen, who was in the wards of my esteemed colleague, Dr. Carl Potain, presented us with an example of these singular phenomena. His father had on several occasions manifested suicidal tendencies; his mother was said to have been subject to convulsive attacks, perhaps of epilepsy, but at the very least of hysteria; and his first fit, which had occurred when he was eleven years old, had been caused by the deep impression made on him by his mother's decease. On the accession of every fit, which now returned frequently, this painful circumstance invariably recurred to his mind. "*I am seized through my thoughts,*" he used to say, and he explained to us that his thoughts were always the same, and constantly referring to his loss.

Epileptics usually remain after their attacks for a length of time varying from a few minutes to several hours, in a state of more or less marked torpor or semi-hebetude. They have a difficulty in co-ordinating their ideas, in recognizing the persons or objects around them, and their mental confusion, especially the failure of their memory, lasts for one or two days. But if this be the usual state of things, it does not infrequently happen that this perturbation of the intellect, after having expressed itself by stupor and prostration more or less prolonged, suddenly manifests itself by cerebral excitement, by a furious delirium which prompts the unfortunate patient to the commission of acts of the most violent character, so much so, indeed, that no madman, as everybody knows, is more vicious or more dangerous.

"No one," says the author of the excellent memoir which I recommend you to read, "no one can form an accurate notion of the sort of rage which suddenly possesses the epileptic, and drives him to strike or to break anything which he can lay hold of. During these transient attacks of furor, he is so dangerous to those around him, as well as to himself, that the attention of persons in authority and of medical men cannot be too earnestly drawn to these conditions of instinctive and blind violence which all authors have pointed out as frequent results of epileptic fits. They may lead to the infliction of grave wounds, to the commission of suicide, of homicide and arson, and yet the individual cannot be held responsible in any degree for the acts of violence perpetrated by him during this perfectly automatic though short-lived delirium.[1] In a former lecture on apoplectiform cerebral congestion, I related a few instances of this kind. I need not

[1] Jules Falret, *loco cit.*, p. 967.

revert to the subject, but will merely add the following case, which many of you will doubtless remember :—

At the end of December, 1860, a young woman was admitted under my care into the St. Bernard ward, in a state of wild delirium, which was said to have commenced a few hours previously. I told you at the time that she was epileptic, and on the next day her husband communicated to me some important facts which entirely confirmed my diagnosis. He told me that his wife had suffered from epilepsy for more than a year, and that on the day preceding her admission into the hospital she had been seized with transient vertigo, followed by wandering for a few minutes. During the night she had a severe epileptic fit, after which the delirium had set in. This attack lasted five or six days.

"In some cases the delirium, which may last a few hours only, persists for twelve or fifteen days, although it generally passes off after two or three days. In some individuals the temporary intellectual disorder which succeeds an epileptic fit does not show itself in its usual form of instinctive, blind violence, but assumes the form of more or less marked simple maniacal excitement. The patient talks incessantly and incoherently. He moves about restlessly, and executes movements that are more disorderly than violent. He is sometimes under the influence of delirious ideas of an agreeable nature, which rapidly alternate with conceptions of a painful kind, and frightful hallucinations, chiefly of vision. But this temporary maniacal delirium consists in a rapid succession of incoherent thoughts, and in great disorder of actions, rather than in extreme violence, as is on the contrary observed in the class of patients we spoke of before." [1]

I now pass on, gentlemen, to the consideration of the morbid psychical phenomena, which, in the division I borrowed from Dr. Falret, are comprised in the third category. They are those intellectual disturbances which occur either in direct connection with convulsive and vertiginous symptoms, or independently of them, in the form of more prolonged attacks, and deserve more especially the name of *epileptic insanity*.

A detailed description of these phenomena is of such vast importance to the practitioner, that I will quote in full the following extract from Dr. Falret's memoir :—

"Two forms of well characterised intellectual disturbance, constituting genuine attacks of insanity, may occur in epileptics at various intervals, and as irregularly as the convulsive seizures themselves. They are sometimes directly connected with those seizures; but may at other times be independent of them.

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[1] Jules Falret, *loco cit.*, p. 697.

They are often confounded together in a common description, but they deserve to be described separately, in spite of the points of resemblance between them. In order clearly to distinguish one form from the other, we shall give them names which will have the peculiar advantage of recalling the striking analogy which exists between them and the two kinds of seizures which authors have pointed out. We shall call one form *petit mal*, and the other *grand mal;* meaning thereby to indicate the close relationship observed between the physical and the mental manifestations of epilepsy."

"*Petit Mal.*—The patient suffers at intervals from a more marked intellectual disturbance, which lies midway between the slight degree of impairment characteristic of his habitual state, and the attacks of furious mania of which we shall speak presently. This intellectual disturbance, the duration of which varies from a few hours to several days, recurs in paroxysms. It consists principally in a great confusion of ideas, accompanied in most cases by sudden instinctive impulses and by acts of violence, phenomena entirely special to epileptics, and intermediate between the mental lucidity of partial delirium and the complete disturbance of general delirium.

"Epileptics subject to this particular form of delirium generally become at first sad and morose without cause; then suddenly get into a state of great despondency, attended with obtusion of ideas and feelings of irritation against everything around them. They feel somewhat giddy, they say; they are partly conscious of the vagueness of their ideas, of the failure of their memory, of the difficulty they have in collecting their thoughts and in fixing their attention, as well as of their involuntary violent impulses. The majority of them have in addition, from the beginning of the attack, a deep feeling of their inability to resist a superior force which holds their will in subjection, and drives them, in spite of themselves, to acts of violence. They express this feeling differently according to their education and social position; but in nearly every case analogous expressions are used to describe the same inward feeling. They say, for instance, that they are no longer themselves; that the disease drives them on; that they have within them an evil spirit which commands them, &c. They all, in one form or another, speak of their will being driven on, a circumstance which seems to be a characteristic feature of this form of delirium, and which persists, to a variable degree, during its entire duration.

"Under the influence of this mental condition, such patients suddenly cease their occupations or leave their homes and wander here and there in the streets or in the fields. This *impulsive want to wander about* is nearly constant in this mental state, and deserves to be particularly pointed out. The victims of a vague

sense of anxiety, of an instinctive and groundless terror, of a want of automatic and undetermined motion, these unfortunates feel sick of life, and wander about without any aim or object in view. In their mental confusion they recall to memory all the painful thoughts which they have had at various periods of their lives, and which spontaneously recur to them unchanged whenever they are attacked. They feel intensely miserable. They believe themselves to be victimised and persecuted by their relatives or their friends. They accuse all those with whom they have been in contact of being the cause of their trouble. If they have previously harboured any feelings of hatred or thoughts of revenge against any one, these feelings are quickened by their complaint, and suddenly roused to a pitch of intensity which prompts them to immediate action. The *essentially impulsive and spontaneous* character of the epileptic delirium is really very remarkable. In this state of extreme mental disturbance, of general anxiety and instinctive impulses, the patients are apt, in a most sudden and unexpected manner, to commit all kinds of violence—suicide, theft, arson, and homicide. Some, in order to escape their inward anxiety, attempt to commit suicide; others, under the influence of a similar despair, and of a similar desire to escape their intolerable inward sensations, knock their heads against walls; or, seizing hold of the first instrument they can, strike, or break everything around them, and thus exhaust their rage on inanimate objects. Others, again, rush with fury on the first individual they meet, strike him repeatedly, and, if others come to his help, strike them also. This circumstance, namely, that *repeated blows are struck, and several wounds inflicted, or several persons injured,* deserves to be especially noticed, in our opinion, and seems to us to *characterize the condition of furor epilepticus;* hence it may be of considerable importance in a medico-legal point of view.

"Immediately after the commission of an act of violence, epileptics subject to this form of delirium may get into one of two moral conditions widely differing from one another. In some cases, what they have done eases them as it were, and at once puts an end to their undefined anxiety and their mental confusion. They are like drunken individuals who suddenly become sober again; they partially recover their consciousness, and begin to understand, although very imperfectly, the gravity of their act. In other cases, they continue to run forwards in a state of great excitement and general disturbance—a state in which they are only very imperfectly conscious of the act which they have just committed, or even retain no recollection of it. *The very great confusion of the memory, amounting almost to complete forgetfulness of a great number of facts,* is therefore, in both cases, an almost constant symptom of this kind of delirium.

"When the patients recover themselves, either immediately after the act of violence which forms the crisis of their attack, or after a certain length of time, they sometimes succeed, by dint of exertion, in recalling to mind many details of the facts which occurred during their seizures, especially those which happened towards the close; but their recollections are always very indistinct. This indistinctness has been erroneously regarded as simulated; but it is perfectly real and characteristic of this mental condition. The epileptics are then in a state comparable to that which succeeds a painful dream. The principal circumstances of the attack have at first escaped them. They begin by denying the facts imputed to them; but by degrees they remember certain details which they at first seemed to have forgotten. On the whole, however, they recollect the facts very incompletely.

"*Grand Mal.*—In all asylums there are found epileptics subject to this form of delirium, which we shall call the *intellectual grand mal*, and which is generally known under the name of *furious mania*. All authors have noted the extreme violence of individuals suffering from this particular form of mental disease. Several of them have even pointed out some of the characters which allow of a distinction being made between this and other analogous maniacal conditions. We have no intention of describing it here in detail, but will only indicate its chief distinctive characters. Thus, a character special to epileptic mania is the *greater rapidity of its invasion compared with that of other forms of mania*. Sometimes, in fact, it is preceded by no premonitory symptom whatever. In other cases there are some physical prodromata—such as cephalalgia, vomiting, injection or brilliancy of the eyes, alteration of the voice, slight convulsive movements of the face or limbs; or mental symptoms consisting in sadness, irritability, or slight excitement. But these prodromata precede at the most for a few hours only the explosion of epileptic mania in its most violent form. Another equally important character of epileptic mania (common, after all, to most intermittent kinds of mania) is *the absolute resemblance of all the attacks in the same patient; not only on the whole, but even in every detail.* When the various phases of a first attack of epileptic mania are carefully observed, one is really struck with the fact that the same patient expresses the same ideas, utters the same words, performs the same acts—in a word, goes through the same physical and moral phenomena on the occurrence of every fresh seizure. His ideas, his language, and his acts are fated, as it were, and recur with surprising uniformity whenever he is attacked.

"During these paroxysms, epileptics manifest most of the psychical phenomena which characterise the maniacal state in general. Their ideas succeed one another with great rapidity.

They talk incessantly. They pass without interruption through the most varied series of ideas, and their acts are as disorderly as their language is incoherent. A peculiar feature of their agitation, noted by all authors, consists in the excessive violence of their acts, which violence prompts them to strike and break with a kind of rage all surrounding objects—to bite, tear, and cry without ceasing—and to knock their own heads with violence against the wall. This state of agitation, which passes on to furious excitement, is sometimes carried so far that such patients constitute the most dangerous class of madmen, are universally dreaded in asylums, and can be restrained and protected only by the most coercive measures—such as the strait waistcoat, or lengthened confinement in a cell.

"But extreme violence is not the sole characteristic which distinguishes epileptic mania from other maniacal conditions. An equally remarkable fact is *the terrifying nature* of their predominating ideas, and *the frequency of hallucinations* of a similar kind to which they are subject—hallucinations of hearing, of smell, and particularly of sight. They have visions almost constantly: they see frightful objects, ghosts, assassins, armed men who rush on them to kill them. They constantly see luminous objects, flames, fiery circles; and a circumstance worthy of note is that the sight of blood and red colours frequently predominates in their visions. These attacks of mania, again, present another very important peculiarity. In spite of the disorder and violence of their acts, *their language is, in general, considerably less incoherent than that of many insane individuals.* It is surprising how easily, in spite of their state of agitation, one can follow the train of ideas expressed by epileptics. Their delirium is more connected and comprehensible than is usual in mania. They understand better the questions that are put to them; they answer them more directly, more exactly; and notice what goes on around them more frequently than most insane persons suffering from general delirium with excitement. The less marked incoherence of the delirium, and the greater distinctness of ideas during the attacks, are all the more remarkable that they singularly contrast with the nearly total obliteration of all recollection of the fit after it is over—a defect of memory which is also an almost constant symptom of the attacks of epileptic mania.

"Before concluding this rapid enumeration of the principal characters which distinguish epileptic from common mania, let us add that the attack generally lasts a few days only; and therefore less than in the other forms of mania. Lastly, *its termination is in general as sudden as its invasion.* In a few hours, sometimes even in less time, these patients return to their normal condition. Scarcely ever do they in some cases remain for a short time in a state of slight stupor, or of physical and

moral torpor, before they regain their reason completely. They recover from their attacks like a man who wakes up after a dream or a painful nightmare; and they have scarcely any recollection of what has occured during their seizure."[1]

These two forms of epileptic delirium—the *intellectual petit mal* and *grand mal*—although presenting differential characters, as distinct as those we find in cases of insanity, between partial and general delirium, have also many points of resemblance which denote their common origin. In both the delirium comes on in paroxysms of relatively short duration when compared with those which characterise other mental diseases. Its explosion is sudden, its disappearance no less so; and, after it has passed off, the patient has totally, or almost totally, lost all recollection of the ideas which have passed through his mind, and of the acts which he has committed—of his painful thoughts, his frightful hallucinations, and his instantaneous acts remarkable for their extreme violence.

The identical nature of these two varieties of epileptic insanity is proved, first, by their frequently occurring alternately in the same individual; secondly, by the fact that either in the same or in different individuals, a great many intermediate conditions may be observed, varying from a simple transient cloudiness of the intellect up to the most furious maniacal excitement; and thirdly, by the more or less direct and immediate connection, in the case of *petit mal*, with attacks of vertigo, and in that of the *intellectual grand mal* with the convulsive form of epilepsy. The intellectual impairment increases in proportion to the number of epileptic seizures, the rapidity with which it sets in depending on the frequency of the fits, for the first period of the disease is almost always free from delirium, this happening more frequently during the middle period, that is to say, when for some years already there have been manifestations of epilepsy, at more or less distant intervals. In the last period, when the attacks have recurred frequently and for a long time, the patients fall by degrees into a continuous condition of dementia and idiocy, only interrupted from time to time by phases of agitation of short duration.

This dependence of intellectual deterioration on the duration of the disease and the frequency of recurrence of the attacks, explains how it happens that all ages are liable to mental failure.

I lately saw a remarkable instance of this in a child aged four years and a half. He had been epileptic since the age of 18 months, when he had first presented vertiginous symptoms, con-

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[1] Jules Falret, *loco cit.*, p. 671, *et seq.*

sisting in a kind of hebetude, of bewilderment, which suddenly came over him, and lasted a few seconds. In the space of two months he had five or six attacks, and, after passing a year without any, he became subject, when three years old, to convulsive paroxysms, and to attacks of vertigo, recurring at intervals. When I saw him he had, for the previous three weeks, been frequently seized with convulsions, and the vertigo was almost constant. In the intervals between the fits his reason was impaired; he uttered savage cries, spoke incoherently, and he often bit the persons who waited on him, not excepting his mother.

In consequence also of this dependence, to which I attach great importance, we can understand why, in cases of epilepsy occurring late in life, insanity may not be brought on by it. Calmeil has recorded, however, the case of a woman, aged 73, who became insane after a first attack of epilepsy. The reason is, gentlemen, that like the physical phenomena of epilepsy, its psychical manifestations present the same diversities in their course, their frequency, and the order of their sequence. Thus, in some cases—but very rarely indeed—the convulsive or the vertiginous attacks are invariably attended with delirium; in others, and this is what more frequently happens, the convulsions or the vertigo are alone present; in a third class of cases, again, paroxysms of mania alone attract attention, whether these occur in the intervals between the attacks of *grand mal* or *petit mal* in known epileptics, or in individuals whose complaint is unknown, as in cases of nocturnal epilepsy for instance; or lastly, whether they affect persons who, at the time of observation, have not for a long period been seized with convulsions or vertigo, in consequence of a real transformation of the disease.

If it may be stated as a general law, that epileptic attacks recurring frequently, and over a long period of time, bring on as a consequence an absolute impairment of the intellect, the last term of which is dementia and idiocy, you will meet, however, with epileptics who, in spite of the intensity and frequent recurrence of their attacks, preserve their faculties in all their integrity, and present only slight perturbations of the intelligence and of the temper which cannot be termed insanity. Then, also, by the side of patients whose paroxysms of delirium return at very short intervals, you will see others whose mind is perfectly sound, and is disturbed only by very few attacks, separated by very long intervals, or, perhaps, by a single attack only throughout their whole life.

Setting aside exceptional facts, I shall now conclude what I had to say on this important question, with another quotation from Dr. J. Falret's memoir:—

"The most favourable conditions for the production of delirium are the following:—

"When the disease has been for a long time suspended, it often bursts out with fresh intensity, both in the convulsive and the delirious form.

"When the fits recur at very short intervals, in a series, and as it were one upon the other, delirium frequently sets in, especially when the seizures are imperfect, incomplete, when the *disease does not find a vent*, according to an expression used by the patients themselves and by their friends. Thus, in our opinion, can we reconcile the two apparently contradictory opinions expressed on this point by several authors who have especially studied this subject.

"Delasiauve, for instance, thinks that 'maniacal symptoms are more likely to show themselves, in proportion as the fits recur at shorter intervals, more frequently, and with greater intensity, and in proportion to the duration of the disease.'"

On the other hand, Morel[1] says:—"I have noticed that epileptic fits are complicated with exaltation, which is more marked in proportion as the attacks are separated by longer intervals, and as the individuals enjoy their reason more completely during those intervals." In the next page, Morel declares that he also adopts Dr. Cavalier's opinion, touching the greater influence of imperfect epileptic attacks on the production of delirium.

These opinions, which are apparently contradictory, may, however, we believe be included in the following proposition:—*Delirium chiefly occurs as a consequence of epileptic attacks recurring at short intervals, after a prolonged suspension of the disease.*[2]

§ 4.—On hereditary taint, as a predisposing cause of Epilepsy.—Influence of Marriages of Consanguinity.

In a former lecture, I mentioned some of the reputed exciting causes of Epilepsy. I wish now to draw your attention to its most powerful predisposing cause.

Hereditary taint has certainly a great influence on the production of epilepsy, and I hardly understand how trustworthy authors can have doubted such a fact, which has been accepted by the generality of practitioners. They may have been misled by the circumstance that disorders of the nervous system assume the form of epilepsy in some individuals, and in others of phenomena of an apparently different character. This *transformation of nervous* affections into one another is a vast subject, which I cannot consider now; but if you question your patients scrupu-

[1] Morel: "Etudes Cliniques," t. 11, p. 319.

[2] Consult the exhaustive chapter "On the Mental Condition of Epileptics, in its several relations," in Dr. Russell Reynolds's work on Epilepsy, p. 162.—Ed.]

lously, if you carefully enquire into their previous history, you will, in many cases, discover, either in their direct or collateral relatives, symptoms analogous to those which they themselves present, or mental alienation in one of its various forms, or mere eccentricities of character or of manner, or, again, disturbances of innervation characterized by strange symptoms, by peculiar nervous phenomena, which indicate an unfortunate predisposition transmitted from generation to generation.

I will give you a few instances in illustration. The first one which I am going to relate struck me particularly, and from special circumstances I was enabled to study it carefully. A gentleman, now 88 years old, was affected, at the age of 64, with melancholia, of which he is at present perfectly cured. He had three children, two sons and a daughter. The eldest son is of a melancholic temperament, but of perfectly sound mind; the second was affected with locomotor ataxy, and died mad. A son of the latter, at present 30 years old, is as yet of sound mind, but has a child who is an idiot. The daughter, who is devoid of intelligence, and is, besides, somewhat strange in her ways, has had two sons, the eldest of whom died insane and paralyzed, whilst the younger one is almost idiotic.

This gentleman had also a sister who became mad at the age of 30. This lady had a son and a daughter; the first, from infancy, has suffered from night-blindness, and is now afflicted with epilepsy; the second was amaurotic, and died insane, leaving also a son, who has already given proofs of a notable impairment of the intellect.

I was once asked to see a child suffering from epileptic vertigo. His father's intelligence was below the common average, manifestly owing to a defective mental organization, and his mother informed me that a brother of his had for two months been troubled with a strange, convulsive cough, somewhat like whooping-cough, but essentially different from it in many respects. This cough, which had worried him, and incessantly prevented him from sleeping, ceased suddenly after the administration of two granules of santonine, which brought away some ascarides lumbricoides.

Those nervous symptoms, that convulsive cough, were not in themselves extraordinary. They have been long ago pointed out as belonging to the train of morbid phenomena caused by the presence of worms; and, among other instances, some of you may perhaps know the case related by Dr. Graves in his clinical lectures.

A young girl was for several months troubled with a constant cough, accompanied by fever and unpleasant general symptoms. She lost flesh considerably; so much so, indeed, that Dr. Graves and Sir W. Crampton, who saw her in consultation, believed her

to be consumptive, although they never could find any sign of phthisis. The cough persisted; hectic fever and the loss of flesh became more marked; but one day, after having for some little time taken oil of turpentine, which an old nurse gave her, she passed a tape-worm, and was at once cured.

In the case of the boy to whom I just now alluded, the nervous symptoms were not therefore extraordinary; but they indicated an hereditary taint which could not be referred to the father's imbecility, and which in the other boy manifested itself by epileptic vertigo.

Such examples of predisposition to various nervous disorders, transmitted from parent to offspring, abound in the records of medicine; and, among those which have fallen under my own observation, I will mention the following:—

A gentleman, the son of a celebrated painter, and himself an excellent draughtsman, and a pupil of Gros, had to give up painting—or, at least, to confine himself to sepia drawings—in consequence of a peculiar defect of vision, with which he had been afflicted from birth—namely, inability to distinguish red from green. Thus, the red fruits and red flowers in his garden looked to him of exactly the same colour as the grass on his lawn and the leaves on his trees. He was incapable also of seeing the difference between the red ribbon of the Legion of Honour which he wore, and the green ribbon of another order. In all other respects his sight was excellent, so that the defect was as strange as inexplicable, and must probably have been due to a defective organization of his nervous system, although he had never suffered from any nervous complaint. This peculiar defect of vision has, by ophthalmologists, been described under the name of *Daltonism;* and, in his treatise on Diseases of the Eye, Mackenzie has recorded several examples of it.

Now, this gentleman was the father of seven children, six of whom had convulsions in infancy, whilst in one of them, whom I attended for a long period, symptoms of eclampsia complicated attacks of acute catarrh, pneumonia, measles, and scarlatina, from which he suffered at different periods, and showed themselves as well during his rather difficult dentition. A few years afterwards he was seized with well characterized epilepsy, which carried him off at the age of twenty.

Not long ago I had under my care, in the St. Bernard ward, a woman, aged 40, who for the last three years had been subject to epileptic vertigo. Whenever she was seized she ran quickly straight before her, fell down after a few seconds, but was only partially insensible. When she got up she looked stupid, and continued so for several hours. One of her sisters suffers from similar attacks; and her father had such a violent temper that he attempted to kill her with an axe for some trifling cause only

eight days before his death, which was preceded by nervous symptoms.

The hereditary predisposition of an epileptic may therefore be traced merely to strange nervous phenomena, perfectly different from epilepsy itself, whilst similar disorders may alone be manifested by his posterity, direct or indirect. I wish, gentlemen, to draw your attention particularly to this fact—namely, that the hereditary transmission of epilepsy, and more generally of various nervous affections (in fact, as is the case with all hereditary diseases), may be direct or indirect. In a great many cases, for instance, on inquiring into the family history of an epileptic you will find, on the father's or on the mother's side, sometimes (but very rarely) on both sides, either original traces of epilepsy, in one of its various forms, or one of those affections of which epilepsy may be merely a transformation, and into which it may in its turn be transformed; or, again, cerebral diseases—such as softening, hæmorrhage, &c. In other, and perhaps more common cases, you do not find these primitive traces of epilepsy in the parents themselves, but you have to seek for them in the grandparents, in the direct or distant relatives, in the maternal or paternal uncles, aunts, and cousins. The hereditary transmission may have spared a generation, although the disease, at first latent in the parents, may show itself at a later period in them, after the children have been first attacked.

Besides, cannot the same thing happen in epilepsy as in other diseases? Very trustworthy authors state that "individuals, born of a second marriage between a perfectly healthy woman and an equally healthy man, have been seized with the same complaint as the children born of a former marriage, a complaint to which the woman's first husband was subject."

According to Dr. Olgive, quoted by Dr. Boudin, *a woman, at Aberdeen, had married twice, and had borne children both times. All of them were scrofulous, as her first husband had been, although she herself and her second husband were perfectly free from all scrofulous taint.*[1]

Vidal (de Cassis),[2] also cited by Boudin, relates that a woman, whose first husband had suffered from very obstinate syphilis, gave birth to a child, who died, after presenting the most marked signs of syphilis. After the death of her husband, this woman, who was perfectly healthy, married again. Her second husband was perfectly healthy; but, although she knew him alone, she gave birth to a syphilitic child, four years after her first marriage.

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[1] J. Ch. M. Boudin: "Dangers des unions consanguines et nécessité du croisement dans l'espèce humaine et parmi les animaux (Annales d'hygiène publique et de médecine légale." 2e Série, t. xviii. 1862).

[2] "Traité des Maladies Vénériennes"; 2nd ed. Paris, 1855. p. 539.

However inconclusive these facts may be when taken singly, and however strange they may appear, they suggest reflections at the very least, because what happens as a biological phenomenon may occur as a pathological fact, both in man and the various classes of animals. Now it is well known to zoologists (and the experiment has been often repeated in domestic animals), that females do sometimes give birth to individuals which bear a marked resemblance to the males by which they were fecundated on a former occasion. To give you an ordinary instance of this. Many of you doubtless know that it is not uncommon to see puppies resembling, in form or colour, those of a previous litter, and in nothing looking like their father.

As regards the human species, Dr. Nott [1] gives cases of negro women, who, after having borne children for a white man, continued to have mulatto children with a negro husband.

According to Dr. Simpson, of Edinburgh, a young woman, born of white parents, and who had a mulatto brother born before marriage, had undoubted traces of black blood.[2]

Dr. Dyce says that he knew a half-caste woman who had fair children with a European; and who, on being married to a mulatto afterwards, gave birth to children resembling her first husband both in face and complexion.

Whether they be explained by the impression made on the female generative organs when first impregnated, which impression persists even through succeeding impregnations, or whether they be regarded as inexplicable, such facts exist nevertheless, and open up a vast field to the etiology of diathetic diseases, and we should take them into account in our present inquiry.

With this question of the hereditary transmission of disease is connected another which engages the attention of serious men, and is more than ever now the order of the day. I mean *the fatal influence of marriages of consanguinity on the propagation of the species.* These influences play some part in the history of epilepsy, and it behoves us therefore to say a few words on them.

You doubtless know some of the curious and interesting results obtained from statistical researches made in America, Germany, England, and France. From these researches, and especially from those which my learned *confrère*, Dr. Boudin, has recorded in his memoir, it appears that intermarriages may cause either *complete sterility*, or a *greater frequency of miscarriages;* or that they may give birth to children who die in infancy in a greater proportion than those born under other cir-

[1] "Types of Mankind"; 4th ed. p. 806 (cited by Boudin).
[2] "Gazette Médicale de Paris," 16 Avril, 1859; p. 231 (quoted by Boudin).

cumstances, or who are less apt to resist disease if they live beyond the first period of life, or who are of a lymphatic temperament, with a predisposition to scrofulo-tubercular affections.[1] These intermarriages may, again, beget individuals suffering from degenerations, and physical or moral infirmities; from *monstrosities*—such as polydactylia, spina bifida, talipes, hare-lip, as in the cases reported by Dr. Devay, in his *Traité spécial d'Hygiène des Familles*, who adds also retarded dentition as another consequence of the same cause.

In the lower animals *albinism* may almost at will be produced by successive unions between near relatives; and this singular degeneration in man, of which pretty numerous examples are on record, may perhaps have the same origin. *Diseases of the organ of vision* may be produced; consisting sometimes in strange defects of sight, at other times in total blindness, or in that affection described under the name of *pigmentary retinitis*, which is characterized during infancy by a failure of the sight in the twilight, and a diminution of the field of vision by a feeble light; later, at about the age of thirty or forty, by the abolition of vision, or at least of the faculty of guiding oneself, although the smallest type may still be distinguished within very narrow limits of the field of vision. The ophthalmoscope detects, in such cases, grave alterations of the choroid and of the optic nerve; the retina is more or less atrophied, and is covered with cells of black pigment, which unite and form a plexus.[2] In order to prove to you the relation of these morbid conditions to the intermarriages from which spring the unfortunate patients, allow me to quote a few figures from Dr. Boudin's memoir.

"Among the issue of 27 intermarriages observed in America, Dr. Bemiss[3] (of Louisville) found two children that were blind, and six who were afflicted with various defects of vision.

"Dr. Liebreich, of Berlin, thinks that nearly one-half (27 out of 59) of the individuals suffering from pigmentary retinitis are born of intermarriages." Of these 59 cases, retinitis coincided with deaf-mutism in 18, and in 2 with idiocy. This coincidence is all the more striking that pigmentary retinitis is very rare; and, as Liebreich remarks, that both diseases simultaneously attack children belonging to families in which they show themselves together, but never separately.[4]

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[1] Rilliet (de Genève) "Note sur l'influence de la consanguinité sur les produits du mariage" (Journal de Chimie, Médécine et Pharmacie, 20 Juin, 1856), quoted by Dr. Boudin, p. 61.

[2] "Annales d'Oculistique," Avril, 1861 (quoted by Dr. Boudin, p. 55).

[3] [See: "On Marriages of Consanguinity," by S. M. Bemiss, of Louisville, in "Journal of Psychological Medicine and Mental Pathology." By Forbes Winslow, M.D., p. 368, *et seq.* April, 1857.—Ed.]

[4] Boudin, *op. cit.*, pp. 54, 55, 56, 57, and 58.

Of all the fatal consequences of intermarriages, the most frequent is, without doubt, *deaf-mutism*. "The proportion of individuals who are deaf and dumb from birth," says Dr. Boudin, "increases with the degree of relationship between the parents. If we assume the risk of giving birth to a deaf and dumb child in an ordinary union to be represented by 1, that risk will be equal to 18 in marriages between cousin-germans, to 37 in marriages between uncles and nieces, and to 70 in marriages between nephews and aunts." [1]

That hereditary predisposition has a very small share in the production of deaf-mutism is shown by the fact, that the hereditary transmission of the infirmity is the exception, not the rule. Nay, more: "usually, and in the immense majority of cases, deaf and dumb men married to deaf and dumb women have children who can hear and talk. This is, *à fortiori*, true in cases of mixed marriages, that is, when only one of the parents is a deaf mute." [2] Such was Ménière's opinion, whose authority on such matters is not to be disputed.

"Deaf-mutism," says Dr. Boudin again, "is not always *directly* brought on by intermarriages; it is sometimes produced *indirectly* in cross-marriages, in cases of perfectly healthy individuals, free from all infirmity, but one of whom was the issue of an intermarriage."

In support of this statement the author quotes the following case borrowed from a thesis by Dr. Chazarain [3]:—

"Mr. L——, the mayor of C—— (Dordogne) married his cousin's daughter, by whom he had a son and a daughter not only free from all infirmity, but also enjoying excellent health, like their parents. Miss L——, in her turn, married a young man, a few years older than herself, to whom she was not in the remotest degree related, and gave birth to a daughter afflicted with congenital deaf-mutism. The parents of the child reside in a dry and healthy locality, high above the level of the sea, and their means allow them to live in easy affluence. There is no other case of deaf-mutism at C——, nor has there been another case in the family. Lastly, the mother's pregnancy was not marked by anything special."

Now, might not what applies to deaf-mutism in this case be applied also to all the fatal consequences of intermarriages?

A merchant in Paris marries his first cousin; his sister also marries a first cousin, but belonging to another branch. The

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[1] Boudin, *op. cit.*, p. 80.

[2] Pr. Ménière, "Recherches sur la Surdi-Mudité" (Gazette Médicale de Paris, 3e série, t. 1, p. 243).

[3] L. T. Chazarain: "Du mariage entre consanguins, considéré comme cause de dégénérescence organique, et particulièrement de surdi-mudité congénitale." Thèse de Montpellier, 1859.

sister has no children; the brother has three daughters, perfectly formed and in excellent health. Of these three girls, the youngest, seven years younger than the second daughter, has three well-formed children. The second girl has only one, however much she desires to have many; and the eldest, married for more than ten years, remains sterile, to her extreme chagrin.

These facts, especially the last, are of doubtful import, I admit; but were they to be found more frequently, now that careful inquiries are made into everything bearing on the question of intermarriages, they would become of some value, and on this account the most insignificant deserve to be noted.

To conclude this digression, it has never been said that unions between near relatives were necessarily and fatally followed by evil consequences. Medical observation proves, however, beyond doubt (agreeing in this with the experience of legislators, who in a great many countries have, on that account, proscribed marriages of consanguinity), that the bad results which we have enumerated above, are relatively much more frequent in individuals born of intermarriages, than in those born of mixed marriages, and that both in man and in the lower animals which have been largely experimented on; "intermarriages endanger the species through the sterility, the infirmities, and diseases, which may affect the issue of such unions, when fruitful; and in the case of man, such marriages, when repeated during several generations, bring on physical, moral, and intellectual degeneracy, and, finally, the extinction of the family." Such is Dr. Chazarain's opinion, and a great many physicians concur with him.

The fatal influence of intermarriages is a frequent cause of mental diseases. Esquirol, and after him, all writers on mental diseases, have pointed out that in many cases idiocy and mental alienation had resulted from unions between near relatives. Epilepsy is another of these results.

Among others I will relate to you the following instances. I once attended the family of a Neapolitan gentleman who had married his niece. There was no hereditary taint, and yet of his four children, the eldest, a girl, was very eccentric; the second, a boy, was epileptic; the third was of perfectly sound mind; whilst the fourth was epileptic and an idiot.

A friend of mine, who also married his niece, had four children, one of whom was seized at birth with grave convulsions, and another son is epileptic and an idiot.

Not long ago, I saw with Messrs. Moynier an epileptic boy, the son of first cousins; and shortly afterwards I had the opportunity of observing two analogous cases, one that of a young man, aged 32; the other, that of an idiotic child, subject to epilepsy.

Now that I carefully enquire into the question of consanguinity, whenever I see deaf-and-dumb individuals, idiots, and epileptics, I can scarcely tell you how great a share this influence seems to me to possess in the causation of these affections.[1]

§ 5.—Diagnosis between Epilepsy and Eclampsia.—Transformation of Eclampsia into Epilepsy.—Differential Diagnosis from Hysteria.—Symptomatic Epilepsy.—Treatment of Epilepsy.

Of all convulsive disorders, *Eclampsia* is the most difficult to diagnose from Epilepsy. These two affections are frequently confounded together, as I have told you already, and this confusion is unavoidable, if we only take into account the convulsive phenomena which characterize them both. Look, for example, at a woman, seized with eclampsia, in the eighth or ninth month of pregnancy, or during labour; see a child in convulsions, either at the outset of an eruptive fever, or during the period of teething; and however much you may be forewarned, however careful you may be in observing the case, you shall not be able to discover any difference between those attacks and the convulsive form of epilepsy.

Recall to mind an attack of eclampsia occurring in a pregnant woman for instance, who is suddenly seized with convulsions, sometimes after having first uttered a loud cry. Her limbs are distorted, on one side chiefly, her head inclines to one shoulder, whilst her face is turned to the opposite side; her tongue is thrust out of her mouth, and may be wounded, cut, or lacerated by her teeth; froth, tinged with blood, soils her lips and cheeks, exactly as in an individual who has an epileptic fit. The convulsions last from one to two minutes, and are succeeded by apoplectiform

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[1] [See a Critical Essay: "De la Consanguinité," by Dr. Jules Falret, in "Archives Générales de Médecine," Nos. of February, March, and April, 1865.—The following extract from a memoir read at the Academy of Sciences of Paris by Dr. Auguste Voisin, on January the 16th, 1865, shows, however, that this question is far from being settled, and that marriages of consanguinity do not always exert a baneful influence on the issue of such unions: "There are, at present," says the author, "in the commune of Batz (near Croisic, Loire-Inférieure) 46 instances of marriages contracted by individuals already near relatives; 5 by cousin-germans, 31 by the issue of cousin-germans, and 10 by cousins of the fourth degree. From the five marriages between cousin-germans, 23 children were born, none of whom presented a congenital deformity; two of them died of accidental diseases.

"Thirty-one marriages between cousins, the issue of first cousins, produced 120 children, none of whom is affected with congenital disease or deformity; 24 have died of acute diseases. 10 marriages between cousins of the fourth degree gave birth to 29 children, all in good health, except three, who died of acute diseases.

"Only two couples of the 46 have been unfruitful (the man and wife are relatives of the third degree). The 45 remaining couples have had 174 children, of whom 29 have died."—Ed.]

stupor, as in epilepsy again. But we shall be enabled to distinguish eclampsia: first, by the usual recurrence of the seizures, these following one another pretty rapidly; secondly and chiefly, by the circumstances under which the attack comes on; and thirdly, by certain phenomena which precede and accompany the seizure.

Whereas epilepsy, when it assumes the convulsive form very distinctly, recurs at pretty distant intervals—(I, of course, set aside the *status epilepticus*)—at intervals varying generally from a year to six, three, and two months, or a week only: Eclampsia, on the contrary, runs a more continuous course, recurs at very short intervals, and is always imminent so long as the cause on which it depends persists in full force. On the other hand, when once this cause is removed, the recurrence of the convulsions is in general no longer to be feared, whilst a first attack of epilepsy is always a reason for suspecting others, and almost fatally mortgages the future.

In a pregnant woman, for example, or a woman in labour, eclampsia may recur from eight to twenty times in the twenty-four hours; thus resembling, in some measure, the *status epilepticus* I spoke of just now. The patient is not yet out of one attack before she has another, the convulsions beginning even whilst she is still in the state of stupor characterizing the second stage.

The same thing happens in infantile convulsions. The attacks succeed one another rapidly, and when they do not consist in extensive muscular movements, the phenomena described under the name of *inward convulsions*, and which are, to some extent, the analogues of epileptic vertigo, are manifested for two or three days in succession.

There is rolling upwards of the eyeballs, or distortion of the face, or spasm of the respiratory organs, causing momentary interruption of the respiration, which, after a few seconds, goes on with the same regularity as before.

This frequency of repetition and continuity of the attacks are a frequent cause of death, which is then due to the commotion of the nervous centres caused by the convulsions, or to asphyxia brought on by the tonic convulsions persisting for too lengthened a period in the respiratory muscles, and thus interfering with the oxygenation of the blood.

You understand, therefore, how it happens that death is much more frequently an immediate consequence of eclampsia than of epilepsy. It very rarely happens, indeed, that an epileptic is carried off in an attack, setting aside those cases of accidental death, the result of grave and fatal injuries sustained in his fall. It too often happens, on the contrary, that women die of eclampsia, and still more frequently that children are carried off by convulsions.

What I said just now of Eclampsia, occuring in a pregnant woman, or of infantile convulsions, applies equally well to *Saturnine Eclampsia*, and to *Eclampsia depending on Albuminuria*. Nothing, as far as regards the form of the convulsions, distinguishes them from epilepsy. They are pretty distinctly separated from it, however, by the frequent repetition and the continuity of the attacks; this being the rule in eclampsia, and the exception in epilepsy. The latter affection particularly differs also in that, in the vast majority of cases, it strikes an individual in the midst of the most perfect health. Nothing announces the attack; a minute before it occurs, whether preceded by an aura or not, the patient was as well as a week before. This is the rule, and the exceptions to it are much more apparent than real, generally occurring in cases of symptomatic epilepsy, which, strictly speaking, should not be separated from eclampsia.

Eclampsia, on the contrary, only supervenes under certain given circumstances, more or less easily discoverable. It is dependent on a pathological condition characterized by other symptoms, and shows itself at the onset, in the course, or towards the termination of, some acute or chronic disease, and frequently it is even possible to foresee it. Thus Albuminuria, whether idiopathic, or due to Bright's disease or to antecedent scarlatina, or whether occurring in a pregnant woman (in the two latter conditions particularly), makes us dread the possible occurrence of eclampsia. After this has shown itself, independently of the other general or local symptoms which belong to albuminuria, the mere presence of albumen in the urine sufficiently indicates the disease. In some cases, of course, this diagnosis cannot be made, as when individuals, that were previously subject to epilepsy, are seized with eclampsia. Thus again, in cases of convulsions occurring in a child who is teething, or who is at the beginning of an acute febrile affection, you will at once recognize eclampsia, or at least epilepsy will occur to you on second thought only. Yet, gentlemen, some reservation should be made. These eclamptic convulsions, whatever their exciting cause may have been, are often indeed true epileptic fits. It is especially in children above five or six years of age, and even in younger children, that epilepsy may be dreaded for the future, when the attacks of eclampsia occur frequently and for the least thing. I have sometimes also seen epilepsy in women who at some more or less distant period had been seized with eclampsia during labour. I have always asked myself whether there may not be some connection, in such cases, between eclampsia and the epileptic fits, and I have felt inclined to answer the question in the affirmative. These considerations, in cases of infantile convulsions, apply particularly to that form of partial convulsion which affects the

muscles of the larynx, and has been very improperly called *thymic asthma*. You saw an instance of this some time ago in a baby eight months old, whose health was very good in other respects. His mother said that frequently, whether he was at the time sitting in his crib, or was being nursed in her arms, he suddenly uttered a loud cry, as if he felt acute pain. This cry resembled that of a fit of anger, but was immediately followed by a noisy, hissing inspiration, similar to that of whooping-cough. The face was red, the veins of the neck were swollen. After a few seconds, however, the child became calm again, and recovered his previous condition. He had also suffered from regular convulsive attacks.

In such cases—and I shall some day revert to this point, which is of the highest practical importance—be very reserved in your prognosis. Although these symptoms be not serious in general, the patient's life may, however, be in immediate danger when the laryngeal spasm is prolonged beyond a certain period, for, if it lasts two minutes, asphyxia is produced. Moreover, I repeat, these partial convulsions may be a manifestation of epilepsy, which, sooner or later, is attended with more distinct and more characteristic phenomena.

I next pass on to the *differential diagnosis between Hysteria and Epilepsy*. This is, in some cases, attended with great difficulty, as I have already pointed out. Thus, although in general preceded by very characteristic nervous symptoms, a convulsive attack of hysteria may sometimes set in suddenly, or the patient may, at the beginning, have felt a kind of aura, a spasmodic sensation, which, starting from some point of the body, becomes general, and bears some resemblance to the *aura epileptica*. I hasten to add, however, that such cases are exceptional. In the great majority of instances, hysterical convulsions are ushered in by phenomena which, when once observed, can no longer be mistaken. As to the *aura hysterica* itself, it differs widely from the *aura epileptica*. I have already drawn attention to the fact, but it is of such importance that I do not fear to revert to it. The *aura hysterica* starts almost constantly from the same point, and is compared by the patient to the sensation of a foreign body, of a ball pressing on the umbilical and epigastric region, and which, extending upwards along the œsophagus, produces, on reaching the throat, a feeling of choking. However short may be the time during which this sensation lasts, it persists generally much longer than the *aura epileptica*, the rapidity of which may, in almost all cases, be compared to that of lightning.

Another important point is, that hysteria affects the female sex almost exclusively, so that the fact of a patient being a female should put you on your guard, and make you suspect the nature of the symptoms.

The aspect of hysterical persons is besides very different from that of epileptics. As to the attack itself, it is tumultuous in hysteria, more silent in epilepsy. An individual, in an epileptic fit, is convulsed for a few moments, but after a few seconds he becomes motionless and passes into a state of stupor; the deadly pallor of his face is replaced by redness of a more or less livid, bluish tint. Hysterical convulsions are, if I may use the expression, more demonstrative: they consist in extensive movements, which do not affect one side especially, as in epilepsy, but both sides nearly equally, except in cases complicated with catalepsy or paralysis. The patient can be restrained by several persons only. If an epileptic be seized whilst lying down, he remains in his bed; if whilst standing, he falls down, and rarely quits the place where he fell. An hysterical patient, on the contrary, throws herself about in all directions; if in bed, she rises and throws herself to the right and to the left. An epileptic, again, after having uttered the cry which generally precedes a fit, remains silent; an hysterical woman keeps crying during the attack, and goes on moaning, or, towards the close, bursts into tears or into a laugh, without any reason.

Lastly, whilst a fit of *haut mal* rarely lasts three minutes, hysterical convulsions are prolonged for a much longer period.

Such are, speaking very generally, the distinctive characters between an epileptic seizure and an hysterical fit. As to the fundamental differences between the two diseases, I need not insist on them now.

There are cases, however, gentlemen, in which the symptoms observed are really on the confines between the two diseases. You may remember a nurse who was formerly in my female ward, and who is at present at the Salpêtrière. She was certainly hysterical, but her attacks sometimes presented, at the onset, the characters of epileptic seizures. You could see at the same time, and in the same ward, that young girl whose history I have already related to you, and who, during her epileptic fits—which lasted, it is true, one minute only—was frequently agitated with the same violence and the sort of jactitation which belong to hysteria.

Let me add again (and there are pretty numerous instances of this at the Salpêtrière) that some women are at the same time hysterical and epileptic; and, indeed, there is no reason why epilepsy should protect against hysteria, or *vice versâ*.

I now proceed to the consideration of another point as regards the diagnosis of Epilepsy.

An individual, with a tumour of the brain, of tubercular, syphilitic, or cancerous nature, is seized with convulsions—should they be called *epileptic?* A great many physicians will reply in the affirmative, but will add the qualification of *symptomatic*

to the name *epilepsy*. It is true that these epileptiform convulsions differ in nothing from those of genuine epilepsy, but the seizure is sometimes preceded by more or less violent headache, which is exactly localized by the patient. Sometimes also, there exists more or less complete paralysis, limited to one side of the trunk, the muscles of the face, the eyes, the soft palate,—a paralysis of movement to which is, in some cases, superadded a paralysis of sensation; lastly, there may be also impairment of the intellect. Now all these symptoms indicate the existence of some more or less profound organic lesion of the brain.

The following case is a remarkable instance of this, and, although it was one of the first of the kind which came under my observation, and although more than thirty years have elapsed since then, I still remember it perfectly. A gentleman was one evening, for the first time in his life, seized with epilepsy whilst at the British Embassy. Shortly after this he had a second attack, and on one occasion, whilst riding in the Champs-Elysées, he fell down from his horse in a fit, and severely injured his head. From that time he gave up going into society, and consulted Dupuytren, who prescribed, but without success, the remedies vaunted against epilepsy. He next placed himself under the care of Dr. A. Lebreton, who, on carefully inquiring into his previous history, ascertained that he had suffered from violent and chiefly nocturnal headache. I was then asked to meet Dr. Lebreton in consultation, and we together made out that the pain was almost exclusively limited to one side of the head. The periodical recurrence of the headache and its nocturnal exacerbation pointed clearly to syphilis; and, indeed, we ascertained on inquiry that the patient had had a venereal affection five or six years previously, to which he had never paid attention. Suspecting, then, an intracranial exostosis, or a syphilitic tumour, we recommended a treatment chiefly consisting of Liq. Van Swieten.[1] The symptoms disappeared completely from that time, and a radical cure was obtained.

This case, then, was one of epileptiform convulsions, or of eclampsia, to use the expression which is current in the profession, but it was certainly not a case of epilepsy in the sense usually meant.

In some cases the form of the seizure resembles epilepsy still more closely.

Last year I was consulted by a lady 71 years old, who, since the age of 40, had been subject to attacks recurring with a daily

[1 The Liq. Van Swieten is a solution of corrosive sublimate; 1 part in 1000. Dose: ʒj.—Ed.]

increasing frequency, and so much so that she had as many as twenty-one in the twenty-four hours. The diagnosis of her case was written in large type on her face, for she had on the forehead a broad, deep scar, which began above and outside of the right eyebrow, and penetrated the frontal bone, which had necrosed. There had also been necrosis of the nasal bones, for the nose was broken down and depressed.

Under the influence of mercury and iodide of potassium, rapid improvement followed, so rapid indeed that she had only one attack in the very first month, and this proved the last.

In some cases, the lesion, which is the exciting cause of the attacks, is so trifling, that its importance is with difficulty suspected. Dr. Foville saw, with Alph. Robert, my excellent colleague at the Hôtel-Dieu, a young notary's clerk, who, for several years, had been subject to monthly attacks of epilepsy. Many remedies had been tried in vain, when Dr. Foville suggested the extraction of some carious teeth which ached constantly. The suggestion was acted upon, and from that day the fits disappeared.

On March 2nd, 1861, Dr. Monnier, of Saint-Paul (Eastern Pyrenees) communicated to me a no less interesting case, which somewhat resembles the case of Graves which I quoted a short time ago. A man 40 years old, tall and of a robust constitution, was seized, on several occasions and at very short intervals, with violent epileptic attacks. Dr. Monnier, on learning that the patient often passed fragments of tænia, gave him large doses of castor-oil. A whole tænia came away, and from that time the convulsive attacks ceased.

The little success which attended the treatment of epilepsy had, among the ancients, obtained for it the appellation of *morbus sacer*, a scourge sent by the gods in their anger. The unfortunate patient was fatally doomed to convulsions, and nothing short of a special intervention of the gods could save him from the fate which awaited him. The progress of science has little changed matters in this respect, and epilepsy is, in general, as incurable now as formerly. I say in general, and I make this reservation, because there is no medical man, of large experience, who has not seen some epileptics get well. You shall have occasion also to see a certain number of patients remain seven, eight, ten years, and more, without any fresh fits, although these recurred frequently before. Now, in a complaint of this nature, a long truce looks very much like a cure.

When a disease admits of so fatal a *prognosis*, the number of remedies vaunted for its cure increases indefinitely. And as, in some rare cases, a spontaneous cure takes place, the credit is given to the *treatment*, and not to Nature, until repeated failures show the inefficacy of the remedy.

Epilepsy could not escape the common law. Its incurability necessarily led medical men to use against it all the resources of their therapeutic arsenal, so that known drugs, as well as unknown remedies, some apparently rational, others empirical, others again of the most extraordinary character, were tried in succession. And it would be difficult indeed to give a complete list of all the remedies which have in turn been vaunted against epilepsy, and soon justly given up, beginning with those mentioned by ancient authors, some of which were abominable, and invented by superstition, "*quædam satis abominanda*," and "*superstitiosa plurima*," and not forgetting those of which ignorance and bad faith dare exaggerate the virtues, even to this day.

Is medicine, then, entirely powerless against this terrible disease? Not completely so,—for there is a mode of treatment, *the treatment by belladonna*, which, if it cures epilepsy in very rare cases only, procures at least a pretty large number of patients a real alleviation of their sufferings.

Although, from the difficulty we have of judging of its effects, the same objections apply to this treatment as to all the rest, yet scepticism should not go beyond certain limits, and we cannot refuse to believe the testimony of grave physicians. Long ago, according to Murray, Greding had several times administered belladonna—either in the form of powder or of extract—to patients afflicted with simple or complicated epilepsy; and if they did not get well, they improved remarkably at least. These observations were confirmed by Leuret at Bicêtre, and by Ricard; but it is Bretonneau who, in our time, has handled this remedy with the greatest perseverance and success.

Almost simultaneously with the illustrious physician of Tours, Father Debreyne, physician to the Trappe of Mortagne, and a Trappist himself, obtained similar results. As to myself, I have employed it for more than thirty years, and it has seemed the least inefficacious of those I have ever tried or seen tried. Indeed, I can now count a certain number of real cures, and in many cases I obtained an improvement which I dared not expect.

Above all, an essential point must be laid down; namely, that the remedy is to be trusted only in so far as it shall be administered in accordance with certain rules, which should not be infringed. There is a great principle in therapeutics, which should not be forgotten here, less than ever: it is this, that when a disease has deeply penetrated the organism, when it masters its whole substance as it were, one cannot pretend to silence its manifestations, to cure it within a short space of time. A chronic disease requires chronic treatment. Thus, when syphilis dates five, six, eight, ten years back, you cannot hope to

cure it, except on condition of subjecting the patient to a very prolonged treatment, for five or six months at first, and, after a short interruption, resuming it again, and so on for several times. On this condition alone will you succeed in rooting up the evil, and in removing it entirely.

Now, if syphilis requires such prolonged treatment, how much more must epilepsy require it, the germ of which often exists in the system from birth? The treatment should be persevered in, therefore, not for months only, but for several years in succession. The disease is to be allowed no truce, and the system should be kept constantly under the influence of the drug, lest it should be mastered again by the disease which is forcibly kept down. Of this, gentlemen, you should be firmly convinced, and of this you should warn the patient who places himself under your care, and his friends who ask your advice.

Let us see, then, how belladonna should be administered. Pills are made up according to the following formula:—

℞: Extracti Belladonnæ } gr. $\frac{1}{5}$
Pulv. fol. Belladonnæ } āā
pro pil. j; mitte 100 similes.

During the first month, the patient takes one of these pills every day, in the morning, if his attacks occur chiefly in the daytime; or in the evening, if they are chiefly nocturnal. One pill is added to the dose every month, and whatever be the dose, it is always taken *at the same period of the day*. By that means, the patient may reach the dose of from five to twenty pills, and even more. It is impossible to say beforehand what should be the maximum dose; this depends only on the toleration of the drug by the patient, and its influence on the disease. Excessive dilatation of the pupils, and very uncomfortable dryness of the throat, indicate toxic effects beyond which the drug should not be pushed. If the belladonna is borne with very great difficulty, the dose should be increased only every two, three, or four months.

When an improvement seems to show itself, the last dose given is continued for some time, and it is then gradually diminished. Lastly, all treatment is suspended for a time, and is resumed again, after an interval the duration of which should be proportionate to the degree of improvement.

I cannot too much impress upon you that patience, both in the physician and the patient, is the principal condition of success. A year sometimes is scarcely sufficient for discovering the influence of the belladonna; and if in the succeeding year some improvement follows, the treatment is to be persisted in for two, three, and four years, according to the rules I

have laid down, in order completely to master the nervous system.

For some years past, I have used atropia in preference to belladonna. I prescribe it as follows :—

℞ : Atropiæ sulphatis 1 grain.
Spiritûs vini gallici 100 minims.

One drop of this solution, that is to say 1-100th of a grain of atropia, is given instead of one of the above pills, and the dose is increased by one drop for every succeeding month.

Although this treatment, I repeat, has appeared to me the least inefficacious, yet, in the majority of cases, I must confess it, I have seen it fail completely. Belladonna, therefore, is far from being a specific against epilepsy; but it is more valuable than the preparations of silver, of copper, and of zinc, although, when it has proved ineffectual, I sometimes use these with some benefit.

In most cases I combine these various remedies. Thus, I give belladonna in the morning, and nitrate of silver in the evening, ten days running every month. I prescribe the following pills :—

℞ : Argenti nitratis gr. ij.
Pulveris acaciæ } q. s. pro pil. x.
Aquæ distillatæ }

Even to a child, between four and ten years old, two of these pills are given every day.

For the next ten days I replace the nitrate of silver by copper.

℞ : Cupri sulphatis gr. xx.
Sacchari gr. lx.
Misce et divide in pulveres xx

The patient takes at first two of these every day, and he gradually increases the dose to six, always, of course, taking care that the stomach tolerates the drug. In the case of a child, each powder should contain only from 1-5th to 1-4th of a grain of copper.

For the last ten days of the month, I again replace the copper by preparations of zinc, given in pretty large doses. I give the *Lactate of zinc*, associated with sugar, as in the preceding formula, so as to give it in a powder, or in pills made up with confection of roses. The dose is from two to eight grains. After this, I return to the nitrate of silver, then to the copper, and next again to the zinc.

Such, gentlemen, is the treatment which I habitually recom-

mend. You will obtain more favourable results from it in the convulsive than in the vertiginous form of epilepsy. *Petit mal* is indeed considerably more intractable than *grand mal*.

Quite recently, my excellent friend, Dr. Henry Guéneau de Mussy, has stated positively to me that he had been remarkably successful in the treatment of epilepsy by *bromide of potassium*.[1]

On the ground that modifications of the circulation often produce corresponding modifications of innervation, Dr. Duclos (de Tours) thought of treating epilepsy by *digitalis*, which so powerfully modifies the functions of the circulatory system. In a certain number of cases he has seen weekly or monthly attacks diminish in intensity, and even delayed for a period of twenty-seven months. He has also known epileptics thus treated be attacked again only five and even seven years after they had ceased the treatment. He gives the hydro-alcoholic extract of digitalis in pills containing each one grain of the extract. The first day he gives one pill only; the second, two pills: one in the morning, the other at night; on the third day three pills, one in the morning and two in the evening; on the fourth four pills, two in the morning and two in the evening; and lastly, on the fifth day, two pills in the morning and three in the evening. He continues in this way until a sensible effect is produced on the circulation, as generally happens after twelve days or so. He then suspends the treatment for ten days, after which he begins it again, increasing the doses gradually, and then withholding the drug again for some time. He continues in this way for a lengthened period, taking care, in proportion as the treatment is prolonged, to increase the intervals of rest from ten to twenty, thirty, and forty days, ceasing at last after ten months. I have gone into all these details, gentlemen, because the art of administering the drug has an important share in the good results obtained from this method of treatment, and because the physician, who has praised it, is one of the most skilful representatives of the great school of Bretonneau.

I told you that some individuals had warnings of a returning fit in a peculiar sensation constituting what has been termed the *aura*. Cases have been reported by most trustworthy authors, in which the fit was prevented by firm compression applied between the starting-point of this aura and the nervous centres, when the aura began in a limb. Ingenious contrivances have even been invented for facilitating the application of this firm compression. Thus, an instrument-maker made for a young epileptic, who had an aura starting from the thumb, and from there ascending along the arm to the head, a kind of leather bracelet with straps, which could be quickly slipped round the wrist, and tightened with

[1] See the note by the editor at the end of this lecture.

considerable force. I shall not say much of the surgical means employed, some of which appear to me, at the very least, useless. Thus, not only has the *actual cautery* with the red-hot iron been proposed along the course of the nerves which the aura was supposed to follow, but *castration* even has been suggested in the cases where the aura seemed to start from the testicles. Nay more, a singular theory has been broached which has been called the *theory of laryngismus*, according to which epilepsy is said to be caused by occlusion of the glottis, owing to spasm of the laryngeal muscles. Hence, say the authors of this theory, if a passage to the air be opened up, which cannot be closed by the convulsed muscles, all the symptoms will disappear, and they, therefore, have recommended as a "*very simple remedy*" . . . . "*tracheotomy.*" If they do not pretend to cure epilepsy, they at least pretend to ward off the attacks, and to do away with the dangers consequent on them.

I should not have spoken of this savage method, if it had not, of late, had a certain amount of vogue. But whilst mentioning it here, only to stigmatize it, it would be insulting you if I thought it necessary to seriously discuss the subject, in order to prove to you the absurdity of so strange a theory, and the barbarity of a measure which no true physician will be tempted to employ.

ON THE USE OF BROMIDE OF POTASSIUM IN EPILEPSY.

NOTE BY THE EDITOR.

[It is remarkable that Professor Trousseau, who, in his "Traité de Thérapeutique" (Vol. I., pp. 279, 280, 5th edition), has recorded the results of experiments made with bromide of potassium by Mr. Puche, at La Pitié in Paris (as published in 1850 in the theses of Drs. Huette and Ramos), should not have thought of using it against epilepsy, and still more remarkable, that he has not since tried the effects of the drug, after it had been favourably mentioned to him. In this country, however, bromide of potassium has, of late years, been freely administered, since that memorable sitting of the Royal Medico-Chirurgical Society of London, in May, 1853, when Sir Charles Locock, then President of the Society, first recommended its use, on the occasion of a paper on epilepsy communicated by Dr. E. H. Sieveking. "About fourteen months ago," said Sir Charles, "I was applied to by the parents of a lady who had hysterical epilepsy for nine years, and had tried *all* the remedies that could be thought of by various medical men (myself among

the number) without effect. This patient began to take *bromide of potassium* last March twelvemonth, having just passed one of her menstrual periods, in which she had two attacks. She took ten grains three times a day for three months; then the same dose for a fortnight previous to each menstrual period; and for the last three or four months she has taken them for only a week before menstruation. The result has been that she has not had an attack during the whole of the period. I have tried the remedy in fourteen or fifteen cases, and it has only failed in one; and in that one the patient had fits, not only at the time of menstruation, but also in the intervals."

In the beginning, bromide of potassium was only given in cases of epilepsy occurring about the menstrual period; but by degrees, its use became more general; and Dr. Radcliffe declares that "in the summer of 1858, he began to give this medicine almost promiscuously in cases of epilepsy and epileptiform disorder, and from that time to this, he has been continually finding fresh reasons for persevering in the practice." (Lectures on Epilepsy, Pain, and Paralysis, etc., p. 233.) At the National Hospital for Paralysis and Epilepsy, bromide of potassium has been extensively used by Dr. Brown-Séquard, Dr. Ramskill, Dr. Radcliffe, Dr. Hughlings Jackson, and myself. The results obtained are such as to warrant the conclusion that it is infinitely superior to all the other remedies that have been recommended against epilepsy. It is certainly far superior to belladonna (which Professor Trousseau regards as the least unsuccessful remedy) in its power of diminishing the frequency and severity of epileptic fits and epileptiform seizures in general—nay, more, of warding off the attacks, lengthening the intervals between them, and, in some cases, of bringing on a cure. As yet, however, the question of a complete and thorough cure must be left undecided, for time may show that the relief has been only temporary, and that the fits have recurred after a long interval. Many such cases have, indeed, come under my own observation, but in all of them a return to the medicine has brought on another lull of the complaint, and procured another lengthened lease of health. Surely; it is not to be expected that such a tenacious malady as epilepsy can be eradicated at once. In Professor Trousseau's own emphatic words, "a chronic disease requires chronic treatment;" and if bromide of potassium be found capable, as I believe it is, of warding off epileptic seizures for a time, it should even then be regarded as one of the most valuable remedies at our command. The influence of *habit* in causing a recurrence of chronic complaints, after they have been apparently cured, is well known; and in no class of disease is this influence more marked than in affections of the nervous system. May we not reasonably hope, therefore, that by

lengthening the intervals between the fits, by warding them off for a gradually longer and longer period, this influence of habit may be first weakened, next destroyed, and a complete and radical cure be at last obtained. Time and further experience can alone decide; but as yet, the evidence already obtained points decidedly in favour of bromide of potassium.

The *physiological* action of the drug evidently ranks it with contro-stimulants or sedatives, for although it seems to possess a certain amount of alterative power, it is chiefly and pre-eminently a sedative of the nervous system. When given in large doses, such as thirty and forty grains two and three times a-day, it produces very striking symptoms in about ten or fifteen days. The patient at first complains of a dull headache, becomes listless and apathetic, with an expressionless face and a lustreless eye. His intellect is clouded, his mind confused, and he is unable to concentrate his thoughts. There is slowness of perception, and questions have to be asked several times before their meaning is understood, and an answer can be obtained. If, when these symptoms have begun to show themselves, the medicine be continued, hebetude follows, with inability to think and a kind of stupor resembling that of the first stage of typhoid fever, together with drowsiness, somnolence, and constant dropping off to sleep. In no case have I yet seen delirium or hallucinations. The pupils are dilated, and contract very sluggishly under the influence of a strong light; the sensibility of the conjunctiva is so deadened that a finger may be passed with impunity on the surface of the eyeball without producing winking. Hearing loses its usual acuteness, and it is only by speaking in a very loud voice that the patient can be roused from his stupor.

The sense of taste is probably impaired like those of hearing and of sight. The tongue is moist and red at first, but after a few days it has a tendency to drying and browning. There is anæsthesia of the velum palati, the uvula and upper portion of the pharynx, so that these parts may be tickled without producing nausea or involuntary movements of deglutition. Swallowing itself, however, is not impaired, and strangely enough the appetite remains very good; the patient takes his food well, and dozes off immediately after. Digestion seems to be easy, and the bowels, although sluggish in their action, are not very confined. There is intense thirst, and a craving for cold drinks. The anæsthesia is not confined to the mucous membranes only, for the sensibility of the skin is diminished also, so that pinching and pricking are scarcely noticed by the patient. From the beginning, the sexual aptitude fails; erections become rare and imperfect, and cease entirely after a few days.

Simultaneously with the impairment of sensibility, disorders of motility manifest themselves. Thus, the patient is averse to

taking exercise, sits and lounges about; by degrees, his gait becomes altered, he rolls and staggers like a drunken man, his limbs shake and bend under him. After a time, he is obliged to keep to his bed, and when he uses his hands, as in the act of carrying anything to his mouth, they are seen to tremble, as if he were suffering from delirium tremens. The respiration is calm and tranquil, with occasional sighing. The circulation is considerably slackened; the pulse at the wrist is weak and slow, the heart's beat lacking in energy, and its sounds distant and feeble; in fact, in its effect upon the heart, bromide of potassium seems to resemble digitalis. If the drug be withheld, these symptoms gradually diminish and pass off of themselves, but they leave behind them for some time afterwards great feebleness, both physical and mental. The anæsthesia of the fauces seems to be the last phenomenon to disappear. Purgatives and the exhibition of ammonia help in restoring the patient. Dilute mineral acids seem also to be useful in rapidly dissipating these unpleasant symptoms.

Although the exhibition of large doses of bromide of potassium is soon followed by the marked and characteristic phenomena of *Bromism*, it is remarkable how long moderate doses of the drug can be administered without sensibly affecting the system, and producing the above phenomena in a marked degree. Thus, I have seen patients who had taken the bromide for two and three years, in doses of from ten to fifteen grains, two and sometimes three times a day, with occasional short intermissions only, and whose general health had not apparently suffered. At first, from the increase of the appetite, and probably also from the better assimilation of the food taken, the patient gains in size and weight, but I believe that a very prolonged use of the medicine tends in the end to produce a certain amount of wasting. Diminished sensibility, followed by complete anæsthesia of the soft palate, uvula, and upper part of the pharynx, is the first symptom which indicates that the patient is getting under the influence of the drug. The sexual organs are also among the first to be influenced, for there is soon produced failure of sexual vigour, and after a time, marked diminution of the sexual appetite itself. Another frequent, if not constant result of the prolonged administration of the bromide is an eruption of small boils, in successive crops, chiefly over the face and trunk, and accompanied with troublesome itching. From this it may be inferred that the medicine is partly eliminated from the system by the skin, although the kidneys seem chiefly to serve that purpose, the urine always containing a large proportion of the amount of bromide taken. On the system generally, the medicine has a decidedly lowering effect; the patient loses all vivacity, all energy; he becomes low-spirited, indulges in gloomy ideas, and complains of a sense of

intense depression. His circulation is very feeble; his heart is weak, his pulse slow and compressible, the surface of his body is constantly pale, and his aspect in fact is characteristic of anæmia. As to the intellectual functions, they are apparently under the same depressing influence. Ideation and imagination seem to be more affected than the rest, but perception itself is dulled, and memory seems to become less powerful and retentive. But whether these effects are due to the medicine, or whether they are the inevitable consequences of the nervous disorder, of the epilepsy itself—a fearful malady, which entails on its victims failure of intellect, impairment and loss of memory, and but too often idiocy and dementia—it is difficult, if not impossible, to determine. Like all substances endowed with narcotic and sedative properties, and which act specially by lulling the great nervous centres, it is probable, however, that bromide of potassium must, if continued in full doses for a very lengthened period, exert in the end some injurious influence on the keenness of the intellect and the quickness of perception; but between such effects and actual marring and destruction of the mental faculties, there is happily a broad margin. Further experience, however, will settle this, as many other points which are doubtful as yet.

The *therapeutic* effects of bromide of potassium are manifested within a short time. It has a decided and well marked power of checking the fits, and short of averting them, of diminishing their severity and their duration. Under its influence, they become less frequent and severe, the intervals between them more and more prolonged, so that patients who used to have a fit every day, and sometimes several fits in the day, are free from any seizure for a week, and for two, three, four weeks, and more. This influence is extremely marked in recent cases of epilepsy, and seems to diminish in proportion as the disease has extended over a long period of years. In the first class of cases, the intervals between the paroxysms go on increasing in length, whilst in the second, the only sure effect obtained by the administration of the medicine, is a diminution in the number and severity of the fits. As to the class of cases in which bromide of potassium should be given, I believe that it will be found useful in nearly all cases, not only of pure idiopathic epilepsy, but also of what has been called symptomatic epilepsy, as well as in epileptiform disorders in general. Indeed, in all these cases, whether the convulsions be dependent on the presence of a tumor, or of tubercles, or a syphilitic deposit, etc., in the brain or cerebral meninges, or whether they be due to some peculiar change in the intimate structure of the great nervous centres which we fail to detect, their immediate cause is probably some cerebro-spinal modification of which the convulsions are only a symptomatic expression. On this condition it is, I believe, that bromide of potassium exerts its special

influence; but however that may be, an extensive trial of the drug has convinced me of its great usefulness in epilepsy proper and allied convulsive affections. When the disease is traceable to habits of masturbation, the anaphrodisiac property of the drug no doubt explains its favourable influence, and, in many instances (some of which are still under observation), where this cause was suspected to have been at work, I have known the disease kept down, as it were, for four or six months at a time, and even longer.

In one important class of cases, bromide of potassium has failed to do much good, namely in epileptic vertigo. Individuals suffering from a combination of convulsive fits and attacks of *petit mal*, have got rid of the first after a prolonged use of this medicine, whereas the latter have been scarcely modified, except, perhaps, in the frequency of their recurrence and the number of actual seizures. This is another point, however, relating to the use of this remedy, which requires further investigation.

Now, as regards the *mode of administration*, and *doses*, of bromide of potassium. It should be given in doses of from ten to fifteen grains for an adult, at least twice a day; and the best periods of the day are the early part of the morning and the last thing at night. The object of this is to give the medicine when the stomach is empty, as it is more likely then to be absorbed at once, without previous decomposition, free bromine being apt to irritate the stomach. It seems to act better when taken in combination with iodide of potassium and a few grains of bicarbonate of potash, the alterative property of these latter substances helping probably to correct mal-assimilation, imperfect digestion, and flatulency, which are of such frequent occurrence in epilepsy. If symptoms of paralysis have ever manifested themselves, whether lasting or merely temporary, iodide of potassium is more than ever beneficial, and should not be omitted. As the lowering effects of the drug are apt to be very marked after a time, it is advisable to use as a vehicle for it some bitter tonic infusion; or, if plain water be used, to add to each dose twenty or thirty minims of some vegetable tonic tincture. In cases of nocturnal epilepsy, the morning dose may be omitted, and a full dose—say twenty grains—taken at bed-time will be found sufficient.

Even after the fits have ceased entirely, the medicine should be continued in the same doses regularly for at least six or eight weeks, and then one dose alone may be taken at bed-time. It should never be given up suddenly, and it is important that the treatment be resumed after an interval of a month or six weeks, even though in the meantime there has been no threatening of a recurrence of the fits. In fact, the principles laid down by Professor Trousseau respecting the treatment of

epilepsy by belladonna apply equally well to the use of bromide of potassium.

When this medicine has been taken regularly for a long period, the system gets habituated to it, and it seems to exert very little influence, if any, on the fits. In such cases, it should be omitted for two or three months, after which interval it will be found of use again. Incessant pouring in of the drug and soaking of the system with it, as it were, is greatly to be deprecated.

Another important point remains to be considered. Is it better to get the patient at once and quickly under the full influence of the drug—to bring on *bromism*, in a word—or to give moderate doses, and thus gradually modify the system? In a few cases, in which I have seen the first method of treatment tried, I have not known any good results to follow. Hence, until further experience points to the contrary, I am inclined to believe that the administration of moderate doses of bromide of potassium is followed by better results than the more heroic plan of crushing the disease at once by inducing *bromism*.]

LECTURE IV.

ON EPILEPTIFORM NEURALGIA.

The Branches of the Trigeminal or Fifth Cranial Nerve are those generally Affected—The Neuralgia is in Most Cases Accompanied by Partial Convulsions—Is nearly Incurable—Analogy between it and the Aura Epileptica—Differs from Epilepsy, although sometimes Observed in Epileptics—Is Relieved by Section of the Nerve and by Large Doses of Opium.

Gentlemen,—Epileptiform neuralgia presents two varieties. One of these, and the more common of the two, is characterized by neuralgic pain, unattended with convulsive twitches. The other form is accompanied by convulsive movements, and I designate it *tic douloureux*, in order to distinguish it from what is generally and justly understood by *tic*. This latter is a kind of chorea, although in other respects very distinct from St. Vitus's dance, and is a convulsive affection, unattended with pain, which you have often had occasion to see. It consists in rapid, transitory, and involuntary movements of the face, the neck, or the limbs, and which vary indefinitely. *Tic douloureux*, on the contrary, and the non-convulsive form of epileptiform neuralgia as well, always occupy the same seat, or until now, at least, I have only found them affecting the branches of the fifth cranial pair. An individual who, but a moment ago, was perfectly free from pain, is suddenly seized with horrible pain whilst talking. He puts his hand up to his face, and presses it with considerable force, sometimes rubbing it so much and so often that the hairs on that side fall off. (I allude, among others, to the case of that man who has been so long in my clinical wards, and to whose history I shall again revert.) He goes on rocking himself, holding his head between his hands, and uttering half-suppressed groans. This scene lasts for ten, fifteen seconds, one minute at the most, and all is over then without convulsions. The individual resumes his interrupted conversation, until a fresh paroxysm sets in again. This is what I mean by *simple epileptiform neuralgia*.

In another case, simultaneously with the accession of pain, all the muscles of one-half of the face are seen to be thrown into rapid convulsive action, and the attack, as in the preceding case, is over in about a minute. This is *convulsive epileptiform neuralgia*, or *tic douloureux*.

Like everybody else, I used to confound *epileptiform neuralgias* with all the cases in which pain is felt along the branches of the

fifth pair, and which are comprised together under the common appellation of *trifacial neuralgia;* but a few years of practice sufficed for showing me their nature. Whilst the latter were generally of no gravity, and yielded, some of them spontaneously after a few hours or a few days, and others under the influence of proper general or local treatment, I soon found out that the former resisted with a disheartening obstinacy all therapeutic measures, so much so, indeed, that even now, after more than thirty-six years of practice, *I have never known it to be cured in a single case radically.*

I was not long before noticing that this form, which was amenable to no method of treatment, ran the same course as epileptic aura or vertigo, having the same suddenness of invasion, lasting the same length of time, and being especially like them almost incurable. When I compared it with epileptic vertigo, whether or not preceded by a painful aura, and with epileptic fits beginning in one limb and remaining exclusively limited to it, or again with angina pectoris, I could not but be struck with the analogy and the points of resemblance between these various neuroses.

The first case in which I studied this strange neuralgia was that of a man who, in 1831, occupied a bed in the St. Bernard ward, at that time a male ward. I was then physician to the Bureau Central des Hôpitaux, and as such was acting as the substitute of my illustrious master, Professor Récamier. I had the honour of having for my house-physician A. Bonnet (of Lyons), whose premature death science now deplores. This poor patient, who filled some post at the Saint Antoine Hospital of Paris, had for many years been subject to the convulsive form of neuralgia. His paroxysms lasted sometimes a few seconds only, and sometimes a minute; they recurred whenever he spoke, drank, or ate, or whenever one touched with the tip of a finger the few teeth which he had left. The pain was seated in all the branches of the trifacial nerve of one side, but chiefly in the infra-orbital division. Several of the nerve-trunks had been divided already; but the relief had only been temporary, and the pain had always obstinately returned after an interval of from a few weeks to a few months. The extraction of his last remaining teeth gave him no relief. Prolonged applications of a solution of cyanide of potassium did some good. But the pain still returning, as awful and as unbearable as ever, I decided upon dividing the infra-orbital branch. Bonnet performed the operation with great skill; the patient was relieved instantly, and remained free from pain for several months. The following year, I saw him again, suffering in the same way in the course of another nerve of the face, and with the same convulsions. Professor Roux, as far as I can remember, again divided several nerves. Lastly, in 1841,

Dr. Piédagnel saw in his wards at La Pitié this same individual, whom he had known thirty years previously, when house-physician at the Saint Antoine Hospital. The poor man's face was scarred from the surgical operations which he had undergone, for whenever the pain became intolerable, he implored the help of the knife, for this at least gave him relief for a few days, and sometimes a few months.

About the same period I saw in the Marais quarter, a lady 50 years old, who for twenty years had been subject to this *epileptiform neuralgia of the face*. She had from ten to a hundred attacks a day, but sometimes passed a day, a week, or even a whole month, without a paroxysm. The convulsions lasted only a minute at the most, and were confined to the left side of the face; the pain was described as awful. A little relief was obtained by compressing the face with both hands, and this compression, so often repeated during so many years, had produced flattening of the left side of the face. The lower jaw and the malar bone had been, as it were, squeezed down. Dr. Lebaudy divided the temporal branch of the trigeminal nerve, and temporary relief was thus given. But the pain afterwards returned with renewed violence in the other branches which had formerly been less affected. This sad complaint persisted until the lady's death.

In 1846, I saw in my consulting-room a gentleman of about 55 years of age. He had no sooner sat down near me, than he suddenly got up as if moved by springs, and rapidly raising his hands to the right side of his face, which was convulsively distorted, he paced about the room, stamping his foot with a sort of rage, moaning, and groaning like a madman. This strange scene lasted about a minute, and he then sat down. Before he uttered a word, I told him that I knew what he suffered from, and that although I might relieve, I could not cure him. He thanked me for being so candid, and then informed me that he had been, for more than twenty years, subject to this hateful neuralgia, which had always affected the same nerves, and which after disappearing for a few days and sometimes a few months, returned with a hopeless obstinacy, defying the most varied and energetic treatment. Six years afterwards I saw him again; he was still in the same state, for he had refused to try the palliative treatment which I had recommended, and of which I shall tell you presently. At this moment, gentlemen, you can see a similar case in St. Agnes ward. You must have been struck with the look of suffering stamped on his face. Although he is only 48 years old, his face is deeply wrinkled, in consequence of the contractions by which its muscles are almost continually agitated.

He relates that he has always been subject to toothache, but that for the last four years the pain has become so intense that

he has been compelled to consult a medical man. Flying blisters and some pills of which he does not know the composition, calmed the neuralgia for a short time; a year afterwards, he came to Paris and was admitted into Bicêtre. Whilst there he was treated with flying blisters, dressed with morphia. He next went to the Pitié Hospital, for apart from his habitual neuralgia, he had intermittent fever, which was cured by quinine, without the slightest modification of the neuralgia. Eight months later, he was a second time readmitted there, and was treated by my colleague Dr. Marotte. Quinine and iodide of potassium in large doses, blisters dressed with morphia, sulphur baths, faradization, cauterization with the red hot iron of the cheek and forehead, gave no relief.

Two months afterwards, he came here. I at once tried the effect of narcotics in large doses, which in analogous cases had seemed to me to be of great utility. I prescribed for him the aqueous extract of opium, and began almost at once with ten grains taken in the twenty-four hours, gradually and rapidly increasing the dose to *half an ounce*. Within a few days relief was obtained, and four or five months afterwards he felt so decidedly better that he wished to be discharged.

This amelioration did not last long. For three months the man had only a few slight attacks of pain, and he could drink, eat, sleep, and resume his occupation as a copper-turner, but the pain then returned with its former intensity. He was readmitted into my wards, and after being treated in the same way as before, he left markedly relieved.

Last year, however, he returned to the Hôtel Dieu, and was admitted into another physician's ward, where he was treated in the same way again. This time, the pain being less acute than before, the opium had not to be given in as large doses.

Since then, he was free from violent pain; but in April, 1860, the pain having returned with its former intensity, he was for the third time admitted into St. Agnes ward, which he now wishes to leave, feeling quite well again.

With regard to his previous history, he affirms that he has never had syphilis. The only grave disease which he has ever had, is an attack of copper colic, which for a short time compelled him to give up his trade. He also had intermittent fever of short duration. As to his family history, he states that he is not aware of any instance of nervous disease among his relations.

Independently of his paroxysms of pain, he says that he constantly experiences, in the affected side, an unpleasant sensation, which he compares to the oscillations of a pendulum, followed by 7, 8, 10, 15 paroxysms of excessively acute pain, within the space of five minutes. This pain starts indifferently from three constant points, which he indicates perfectly, namely, the points of

emergence of the trigeminal nerve, and is accompanied by spasmodic contraction of the muscles of the face. It is fearfully intense, and drives him to squeeze the affected part violently, and to rub it with a kind of rage. This relieves him a little, but it has been repeated so often that the hair has fallen off from that part. The attacks recur day and night: moral emotions, passing from a warm into a cold place, or the reverse, excite them, and they are more frequent and more violent in damp weather or during atmospheric changes. They are generally accompanied by a more abundant secretion of urine. This almost constant pain kept the poor man in a state of perpetual fear; his intellect, however, has not been in the least impaired, and his memory is perfect. A remarkable circumstance is, that when he has been cured by the prolonged use of opium, he is warned of the return of his attacks by pain in the loins, by an increase of saliva (particularly in winter), and by an eruption of prurigo, chiefly on the back, attended with distressing itching. His neuralgia has always occupied the same seat. His senses are perfect, but reading, if a little prolonged, brings on a paroxysm. Chewing anything hard also brings on an attack. His speech is embarrassed, but it is only because he dares not move his mouth and throw the muscles of the face into contraction, lest he should rouse the pain. His appetite and digestion have been good always.

On this, as on previous occasions, I gave him opium in large doses, and under its influence the same amelioration was obtained. In some cases, the neuralgic pain, after gradually becoming less and less, disappears for two, three, or four months, and when the patient thinks himself cured, returns with renewed intensity, for the space of a few months, and even a year.

Very recently I was consulted by an inn-keeper of Meaux, sent to me by Dr. Charpentier. He was, at the time, subject to attacks which lasted from fifteen to twenty seconds, and recurred every two or three minutes at the most. When they ceased, as they sometimes did for a period of two or three months, he was perfectly cured, for the inferior maxillary nerve, the usual seat of his pain, was completely insensible.

But in the great majority of cases, unfortunately, the relief is not complete, and even when there has been no fresh attack for several months, the patient still complains of a slight degree of pain at the point of emergence of the affected nerve. Whatever be the analogy between true epilepsy and this epileptiform neuralgia, I must admit, however, that the two diseases are merely analogous, not identical; for an individual, subject to epileptic aura or vertigo, rarely escapes an occasional convulsive fit, and it rarely happens, especially, that the intellect be not slightly disturbed during and after the vertigo. Now, in the cases of

epileptiform neuralgia, I have never, as yet, found the least impairment of the intellect.

Still, gentlemen, a few cases that have occurred in my own practice would seem to lead one to believe that, in some cases, epileptiform neuralgia is one of the manifestations of true epilepsy.

I once attended a country practitioner suffering from tic douloureux. For many years, we combated this terrible affection with energy, and in the last period of his life the unfortunate man had genuine epileptic fits.

At this very moment, Dr. Beylard (formerly my clinical assistant) and I are attending together an American gentleman, who, for more than three years, has been subject to awfully painful attacks of epileptiform neuralgia, and to well characterised epileptic fits.

Perhaps, there has been merely a coincidence in these two cases? But were true epilepsy to be oftener met with in connection with this neuralgia, the two diseases should be less separated than I have done, and a kind of relationship should be admitted between them.

I confess that I neglected to inquire into the family history of my patients. But should there be found in this family history, insanity, progressive locomotor ataxy, hypochondriasis, &c., epileptiform neuralgia will, perhaps, have to be placed by the side of epilepsy, and both these affections be looked upon as the expression of one and the same cause. Although from its nature, epileptiform neuralgia may be considered as nearly incurable, I have always thought it my duty to try and combat it by the least inefficacious and the most energetic remedies I had the disposal of. I was besides encouraged by very authentic, although rare, instances in which epilepsy has been cured.

The surgical measures, the utility of which I contested as regards the *aura epileptica*, are sometimes of real service in these cases; and you must at once see the reason of this difference. In the case of an aura, nothing assures us that one nerve is the seat of the sensation instead of another, whilst in epileptiform neuralgia, the seat of the pain can be easily determined. Hence, division of the affected nerves in the points where they can be reached without danger almost certainly gives immediate relief. But I hasten to add that, although I have no hesitation in recommending division of the painful branches of the trifacial nerve, yet I do not expect a lasting good result. Even if I were to see a patient remain better for a pretty lengthened period, I should always dread a recurrence of the disease. I formerly believed, like many others, in the complete efficacy of this measure, but as I grew older, I unfortunately lost all my illusions on that score.

In 1836, Mr. N——, a clerk at the Finance Office, consulted me for an epileptiform neuralgia, which had its starting-point in the tongue. The aura began first in the left half of this organ; from there it spread to the lips, and then to the whole corresponding side of the face, accompanied by horrible pain and by slight convulsions. I tried the most powerful stupefying drugs. Local applications of extract of belladonna and of stramonium, blisters dressed with morphia, the administration of narcotics in very large doses, only produced temporary alleviation; the pain recurred with disheartening obstinacy. I then resolved to divide the lingual nerve, and the operation being somewhat perilous and difficult, I determined to avoid all risks, by proceeding in the following manner:—

I seized the tip of the tongue, taking care to have a piece of linen between my fingers and the painful organ, and passed through it, from behind forwards, a round and curved needle, carrying a silver wire. I next brought the two extremities of the wire together, thus embracing within the circle the left half of the tongue, and I placed them in a knot fastener, which the patient screwed up every five minutes. The first part of the operation was not very painful, and the gradually increased compression produced by the tightening of the knot was attended with much less pain than I had feared. Within five hours the left half of the tongue was thus completely divided, without the least hæmorrhage.

As soon as the compression became a little powerful, all painful *aura* ceased, and the only pain felt was that due to the gradual division of the organ. When the operation was over all pain ceased, and the patient believed he was cured. For nearly a month the apparent cure was maintained, and I was congratulating myself on a success, which in truth I had somewhat expected, when in a short time slight shooting pain attacked the upper lip, on the same side, always retaining the epileptiform character, and attended with slight grimaces and jerks, the whole occurring in less than a minute. A few days afterwards the pain spread to the lower lip, the edges of both jaws, and the infra-orbital and mental branches of the trifacial nerve. Although considerably less intense than before, the pain had not the less returned, and for several years it recurred again. The patient then left Paris, and I lost sight of him.

My excellent colleague, Professor Nélaton, does not simply divide the nerve, but cuts away a portion of it, about one-fifth of an inch. He has often affirmed to me that by this means he had obtained two sound cures. It is true that two years had not yet elapsed when he informed me of his success.

Is it to be said, then, gentlemen, that we can never give relief in such a degree that it may be almost equivalent to a cure? I

confess openly that I have never cured a single patient, none at least of those whom I could see during several years; but I have made the life of some bearable, as you have yourself seen in the case of the individual who is still in my ward, and whose history I related to you.

This is the treatment to which I have recourse; but I must at once tell you that belladonna, which is of some utility against the convulsive form of epilepsy, is almost completely powerless against epileptiform neuralgia, whilst opium procures decided relief:—

An old lady, from Antwerp, placed herself under my care, in 1845, on account of epileptiform neuralgia of the face, to which she had been subject for more than ten years. At first the pain had been slight, and always transitory, affecting one of the divisions only of the tri-facial. Afterwards it had become excessively intense, and had resisted various remedies. The paroxysms lasted from a few seconds to three minutes. Beginning sometimes in the infra-orbital division, and sometimes in the supra-orbital, or the mental, the pain rapidly spread to all three divisions; and when it was at its maximum, it produced spasmodic grimaces of the face. There were sometimes twenty paroxysms in an hour; the least movement brought them on—speaking, coughing, eating, or drinking. In order to diminish the pain she squeezed her face with violence, and moved the skin up and down on the bones. When the pain was more acute, she got up in a sort of frenzy, paced up and down her room, stamping her foot, and uttering muttered groans. This was of such frequent occurrence, that she had become a nuisance to her neighbours, whom she disturbed at night.

The pain disappeared sometimes for eight, fifteen, thirty days, and even longer, but then returned with renewed violence. A remarkable circumstance was, that when the paroxysm was over, the pain ceased entirely, leaving only a sensation of numbness behind.

A good many remedies, rational and empirical, had been tried, but without success. Dr. Sommé (of Antwerp) divided the infra-orbital branch, and thus obtained an apparent cure; but a few months had scarcely elapsed before the pain recurred as before.

After having given her, methodically and perseveringly, some remedies which I thought had not been thoroughly tried, I knew not what to do in presence of so violent and obstinate an affection. I then determined on administering opium internally as a palliative, encouraged in the idea by the fact that I had obtained very evident alleviation of the pain, in this case and in others, by dressing blisters with morphia.

I first gave morphia internally, beginning with pretty large

doses, from 3 to 4 grains a day, and determined on increasing this quantity if the first doses were borne well. I thus came, in less than a fortnight, to administer every day *a drachm* of sulphate of morphia. The amelioration obtained was immense; scarcely were there, in the course of the day, slight shooting pains felt in the branches of the trifacial. Digestion was slightly disturbed; the intellect was normal. But a great difficulty now occurred; the patient's means were limited, and the high price of the morphia almost ruined her. I then had recourse to opium, and in the space of a year she consumed 1,200 francs' worth (£48). This was too much again. The pain recurred whenever she omitted the medicine for eight or ten days, and she was again obliged to diminish an expense which she could not bear. I then obtained of a chemist for her crude opium, at trade price, for which she paid 20 or 25 francs (16 or 20 shillings) a pound. She made boluses of a drachm each herself, and of these she took, according to the pain, from 5 to 20 a day.

It is rather remarkable that these enormous doses of opium did not disturb digestion notably; they caused no drowsiness either, and at night the patient slept as usual. For a period of more than six years I saw this lady from time to time, and I ascertained the following therapeutical results. She was sometimes free from attacks for one, two, or three months; she then suspended the opium, after having first gradually diminished the dose in proportion as the pain itself grew less and the attacks became more distant. On the neuralgia returning of a sudden, with fresh violence, she took at once, and from the first day, as much as 4 and 5 drachms of crude opium, keeping up this dose until relief was obtained. She then diminished it again, because she could no longer take it without feeling nausea and considerable malaise. A few days sufficed for making the pain bearable, I might almost say for curing it, did not slight paroxysms of pain occasionally remind her that she was not cured. By continuing the opium, however, she obtained complete relief for a more or less prolonged period.

Opium, therefore, gave immense relief, but did not cure perfectly; and, I repeat, ever since my attention has been more especially directed to this form of neuralgia, I have never known a case of lasting cure.

It is to opium, then, that I have recourse now, and it is opium which I administered to the patient in St. Agnes ward, increasing the dose, in a few days, as you saw, to ¼ and even ½ an ounce of the extract. But I often meet with patients who dread so energetic a mode of treatment, and with others who, being troubled with vomiting, cannot bear sufficiently large doses.

In the beginning of the summer of 1852 I was, strangely enough, consulted on the same day by two old officers, both subject for many years to epileptiform neuralgia. One of them

was sent me by Dr. Pillon, and I shall relate his history presently; the other by a person whom I had cured of simple neuralgia by a very simple treatment also. The paroxysms returned nearly every 10 minutes, and lasted 40 or 50 seconds. The pain affected the mental and the infra-orbital nerves, and was accompanied by slight convulsive twitches in the whole side of the face. I recommended opium, and prescribed pills containing one grain of opium each, of which four were to be taken on the very first day, the dose to be augmented daily, until the pain was notably diminished as to duration and intensity.

A dose of scarcely 4 grains a day produced considerable drowsiness, nausea, and loss of appetite, but the paroxysms diminished immediately, and the pain became very bearable. I increased the quantity of opium to 10 and even 15 grains a day. The neuralgia was marvellously modified, but the drug disordered the digestion so much, and caused such disagreeable numbness, that I was not able to increase the doses so as to get completely rid of the neuralgia.

With regard to the other case, the following particulars were communicated to me by Dr. Pillon, jun. M. M.——, aged 54, had served in most of the African campaigns, and had suffered from obstinate intermittent fevers, and pretty serious gastric affections; but, with these exceptions, he had always enjoyed good health. In 1845 he, for the first time, felt in the right cheek pain, which was slight in the beginning, and attended with alternate sensations of heat and formication. This pain varied as to the seat of its maximum intensity, this being sometimes about the region of the canine tooth, and at other times about the chin. It lasted from a few seconds only to two or three minutes. By degrees this pain assumed the character which it presented when I first saw the patient. The paroxysms were more or less frequent, but always set in with the same suddenness, making the patient groan from its severity, and clutch the objects near him. All the muscles of the right half of the face contracted with violence, and pulled the features over to that side. After lasting from 12 to 40 seconds, the pain, which had been awfully intense, ceased as suddenly as it had come on. The patient resumed his interrupted conversation, and was perfectly quiet for a period varying from 15 minutes to several hours.

Occasionally the disease assumed a slightly different form. For several hours, several days even, there was no true paroxysm, but slight warnings only, slight shooting pains, which were more frequent in proportion as they were less distinctly characterized. Dr. Pillon, remembering that the patient had suffered from ague, probably caught in Africa, gave him quinine in large doses, but without any benefit. Electricity was employed by Dr. Duchenne (de Boulogne), galvanism by Delacroix; Professor Chomel pre-

scribed Dover's powder, and other physicians recommended Meglin's pills (consisting of valerianate of zinc), valerian, belladonna, cyanide of potassium.

Everything failed.

It was under these circumstances that I saw the patient. At that time the paroxysms had become so frequent, and the pain so acute, that his life was thoroughly miserable. His appetite was failing him, and whenever he endeavoured to take any food, the movements of mastication brought on the most awful pain. The interval between his fits was only of a few minutes at the outside. Dr. Pillon counted as many as seventeen in an hour which he spent with him. His life had become so insupportable that he occasionally thought of committing suicide.

I decided on trying opium in large doses. In the first half of June, the patient took daily from eight to ten grains of crude opium, twelve grains in the second half of the month, and sixteen grains from the 1st to the 15th of August. During the whole of August this last dose was continued, and the paroxysms became very distant, the pain especially very feeble. Life was bearable again, but violent diarrhœa, obstinate cephalalgia and continued nausea, compelled him to give up the treatment. In spite of this interruption, however, the amelioration due to these very moderate doses of opium continued until the end of October. At that time he had only ten or fifteen paroxysms a day, instead of from fifteen to eighteen an hour; and during the night he had three or four only.

These are not excellent results, it is true; but they are favourable upon the whole. Of all the therapeutic agents which I have used—and I have tried a good many with extreme perseverance—opium, then, is the drug which has least disappointed me.

But keep this well in mind, gentlemen, that in the treatment of epileptiform neuralgia, opium should be administered in large doses, which cannot be well determined *à priori*. They should be gradually increased until the pain is quieted, so long as no unpleasant effects show themselves. It may be laid down as a general rule, that the doses which, in a state of health, give rise to very marked functional disturbances, are on the contrary well borne in proportion to the intensity of the pain. There are also idiosyncrasies which cannot be known beforehand, and which may completely preclude the administration of opium in sufficient doses.

Superficial electric excitation has been, in the hands of Dr. Duchenne (de Boulogne), of great service in the treatment of this obstinate neurosis.[1] Almost instantaneous relief is some-

[1] "De l'Électrisation localisée et de son application à la Pathologie et à la Thérapeutique." 2e éd. Paris, 1861, p. 959.

times obtained; but, unfortunately, this important remedial measure fails in the majority of cases to relieve the pain, and to prevent its recurrence.

[Compare, with the above, Dr. C. B. Radcliffe's excellent and original observations on the Therapeutics of Pain, in his "Lectures on Epilepsy, Pain, Paralysis, &c." London, 1864, pp. 309–323.

Dr. Radcliffe speaks positively against the use of narcotics, in the treatment of neuralgic and nervous pain, in doses which destroy the power of feeling pain. He lays great stress on the importance of diet for the cure of neuralgia, recommending a due proportion of oleaginous and fatty matters in the amount of food taken, the avoidance of an excess of sugar, the properly regulated use of alcoholic drinks, the use of coffee, chocolate, or cocoa, in preference to tea, as a common beverage. With regard to medicinal treatment proper, he recommends cod-liver oil, phosphorus in the form of hypophosphites of soda, magnesia, or lime, electricity applied in a certain way, either insulating the patient and charging him with positive electricity, or submitting him to the influence of a succession of shocks from an induction-coil until an artificial hot stage is brought on by paralyzing to a certain extent the vaso-motor nerves. Lastly, he eschews purgatives and aperients, and recommends neuralgic patients to sedulously avoid damp and cold. Under such treatment, and by strictly enforcing the above rules, some of the most obstinate cases of neuralgia have been, he declares, completely and thoroughly cured.—Ed.]

LECTURE V.

ON GLOSSO-LARYNGEAL PARALYSIS.

THERE is a form of paralysis which is always progressive in its course, fatal in its termination, and which is marked, at its onset, by a diminution of motor power in the tongue, the soft palate and the lips. I give to this affection the name of *glosso-laryngeal paralysis*, in order thereby to indicate the principal symptoms which characterize it.

This is certainly not a new disease, and it must have been observed several times already; but as was the case with muscular atrophy, exophthalmic goître and locomotor ataxy, it was confounded with other analogous affections. In 1841, after seeing a patient in consultation with Dr. Vosseur, I wrote a memoir which Dr. Vosseur preserved, and communicated long afterwards to Dr. Duchenne (de Boulogne), who kindly returned it to me. This memoir proves most peremptorily that I had well observed this variety of paralysis, but that case, to which I had not been able to add another, had remained a dead letter for me.

The memoir was as follows:—

"We find that Prince M—— is unable to speak and to articulate any other letter besides the letter *a*; moreover the extreme difficulty which he has in swallowing immediately drew our attention to the organs of phonation and deglutition.

"We ascertained, first, that the soft palate is motionless and does not contract even when directly excited; the tongue moves with difficulty, and the patient cannot curve its tip upwards, and can scarcely protrude it between the teeth.

"When a finger is passed down into the throat, no swelling or tumour is found at the upper part of the larynx. The introduction of the finger gives pain, but whilst the larynx is carried upwards spasmodically by its extrinsic muscles, the pharynx itself does not contract very manifestly. We thought that there was no laryngeal phthisis in the sense usually understood by this word.

"The preservation of the principal vocal sound *a* and its extreme distinctness, indicated that the vocal cords were unaffected. The inability to pronounce the four secondary vowels was solely and perfectly explained by the lesion of the vocal apparatus external to the larynx; just as the inability to pronounce consonants was accounted for by the affection of the

tongue and lips, parts which are chiefly concerned in the formation of these sounds."

We summed up our opinion by saying: "The undersigned think that all these functional disorders are due to weakness of the muscles of the pharynx, the larynx, the soft palate, the tongue, the lips, and the cheeks.

"Similar weakness exists in a very marked degree in the left arm; is a little more pronounced in the left than in the right side of the face; considerably so in the diaphragm, and only slightly marked in the abdominal muscles, the bladder and rectum.

"The consultants have thought that there existed in the nervous centres, and perhaps in the nerve trunks, such a modification that the influx was no longer normally and sufficiently distributed.

"They have asked themselves what this modification could be, and it has seemed to them easier to say what it was not, than to state precisely what it consisted in. They have thought that there was neither chronic softening of the nerve substance, nor effusion of blood, nor a tumour, and they have felt inclined to admit a lesion of the same nature as those which so often give rise to amaurosis, to paraplegia, or to facial paralysis, lesions which dissection cannot always discover or determine."

Surely, gentlemen, we had well seen that this case was a form of paralysis which had not been described in books, and this paralysis was the same which, twenty years later, Duchenne taught us how to recognize.

We had noticed that the patient could only pronounce the letter *a*, and that the vowels *o* and *u* could not be articulated in consequence of the feeble contraction of the orbicularis oris. We had also noted the paralysis of the tongue, the soft palate and the larynx, as well as the great difficulty of deglutition which existed, and we summed up our consultation by saying positively that the functional disorders were due to weakness of the muscles of the pharynx, the larynx, soft palate, tongue, lips, and cheeks.

Far from me is all idea of claiming any priority as to the discovery of this new morbid species. I had seen it, but not seen it with its special characters, and I had soon forgotten it. Perhaps I might have remembered the case of Prince M——, had other similar cases come under my observation. It is just, however, to observe that in Prince M—— I had noted symptoms of paralysis, with progressive tendencies, which have not been mentioned in Dr. Duchenne's memoirs, and which subsequent observation has led me to regard as the fully-developed expression of this disease. Besides, we shall see by-and-by that all these symptoms have a common bond of union, and originate from the same lesion which has certainly its seat in a portion of the nervous system.

But before I give you a general description of this disease, I shall first relate the cases the symptoms of which, will, when analyzed, serve as the basis of my description. Some of you may still remember that woman who was admitted into the St. Bernard ward, No. 29, and whose progressive paralysis, dating from October, 1859, terminated in death in January, 1861. She was forty-seven or forty-eight years old, and a year before coming under my care she had been treated by Dr. Duchenne. She had first noticed that she pronounced some words badly; swallowing next became painful; saliva constantly dribbled out of her mouth; her voice had a nasal resonance; her lips could no longer contract so as to allow her to give a kiss, to whistle, or pronounce the letters *o* and *u*; and, lastly, a few days before admission, aphonia had supervened. When I saw her for the first time in the beginning of November, 1860, I at once observed all the signs of the special progressive paralysis which Dr. Duchenne had just described in the *Archives Générales de Médecine*. There were almost complete aphonia, considerable weakness of respiration, and extreme difficulty of deglutition: so much so, that one day the patient was nearly choked through the bolus of food stopping on a level with her larynx. The progressive paralysis gradually became worse, respiration grew feebler from day to day, and the patient apparently died of slow and prolonged asphyxia. On making a *post-mortem* examination, no appreciable material lesion could be detected in the muscles the functions of which had been principally disturbed, not even with the aid of the microscope. But it is to be regretted that the roots of the hypoglossal nerve, of the spinal accessory and of the spinal nerves, were not examined under the microscope, especially as we already knew the researches of Dr. Duménil in a complicated case of paralysis of the tongue and progressive muscular atrophy. When the *post-mortem* examination was made, however, no modification was found in the size and colour of the roots and branches of the hypoglossal nerve. Still I could not help thinking that there must have been some anatomical lesion of the nervous system, since there manifestly was none of the muscular tissue.

In September, 1862, a compositor, aged seventy-two, was admitted into St. Agnes ward, No. 23. He was of a robust constitution, and had always enjoyed good health until March, 1862. At that time only, he first noticed some defect in his pronunciation of certain words, his tongue felt embarrassed, his voice was altered, and his speech was thick. Exactly as in double facial paralysis, the food lodged on each side between his cheeks and his teeth, and he was obliged to use his fingers in order to replace it on his tongue; occasionally also his voice had a nasal resonance.

In June, 1862, these symptoms having become more distinctly

marked, the patient requested to be admitted into the Hôtel-Dieu. He was at first taken into Professor Rostan's ward, where I had occasion to see him for the first time. The difficulty he had in answering my questions, as well as the evident paralysis of his tongue and lips, reminded me not of the case of Prince M——, for I had forgotten it, but of the woman who had been under my care in 1860.[1]

When this man tried to speak, he uttered a sort of grunt; he could not pronounce a single word distinctly, still less construct the simplest sentence, and although his intelligence was unimpaired, he answered only by signs. His face was expressionless, owing to the immobility of its lower portion and to his mouth being almost constantly open.

On ascertaining what sounds he could utter, I found that he could still articulate the vowels *a*, *e*, *i*, but was unable to say *o* and *u*, for which the lips are indispensable. Nor could he articulate the consonants *p*, *b*, *m*, *n*, *k*, *c*, *t*, which require more or less the intervention of the lips and tongue, as every one may satisfy himself by slowly pronouncing them. The other letters of the alphabet could be articulated, only however on the patient making efforts and pinching his nose so as to close the external nares, and send through the mouth the whole column of air expelled during expiration. The lips, when watched attentively, were seen to remain motionless during the attempts at articulation, at whistling, pursing up his mouth, or pronouncing *o* and *u*. The orbicularis oris did not contract any longer, so that the lips remained half-open. Every moment the patient caught in a handkerchief the saliva which he was unable to swallow, and which his lips could not retain inside the cavity of the mouth.

If he was made to laugh, his mouth afterwards remained wide open, his face looked like one of those masks used by the ancients in comedy, and he was obliged to bring his lips close together again with his fingers so as to close his mouth, and even then he succeeded imperfectly only.

The tongue itself had in a great measure lost its mobility, and was lodged behind the lower row of teeth. It could not be protruded outwards, nor moved sideways, nor raised upwards to the hard palate; it could not be lengthened into a point, nor made hollow in the centre. Its extrinsic and intrinsic muscles, therefore, were paralyzed, and unable to aid in mastication, and assist in tasting the food by pressing it against the roof of the palate. This paralysis of the tongue must also have had a share in causing the difficulty in the first stage of deglutition. As to the paralysis

[1] This case is reported in an Appendix to the *Traité d'Electrisation localisée*, by Dr. Duchenne (de Boulogne). 2nd edit.

of the soft palate, it was proved by the nasal resonance of the voice and by the food getting into the nasal fossæ. The floor of the mouth itself was no longer tense; the larynx no longer rose with the same rapidity during the second stage of deglutition, so that it was probable that the mylo-hyoidei, stylo-glossi and stylo-hyoidei muscles, as well as the levatores and tensores palati, were also palsied. Perhaps were not the constrictors of the pharynx themselves paralyzed to the same degree, for occasionally the posterior aperture of the mouth and the nasal fossæ remaining wide open through the paralysis of the tongue and the soft palate, the food was rejected with violence, as if by a spasmodic contraction of the pharynx. Let us note also that the patient complained of a sense of constriction in the pharyngeal region.

A fact well worth noticing is, that in all the cases which have come under my own observation, the paralysis did not remain confined to the muscles of the soft palate, tongue, and lips, but, after a variable period, extended to other parts of the body, and sometimes showed manifest tendencies to become general. Thus, the compositor whom I mentioned just now suffered from a marked diminution of contractile power in the right arm, which could not be attributed to an old wound. Thus, again, in the cases of Prince M——, of the woman in the St. Bernard ward, and of the man whose history I shall soon relate, the paralysis extended to the chest-walls, the bladder, and lower limbs.

Yet, amidst all these disorders, the intellect was not affected, and the compositor, who could no longer make himself understood by speech or gesture, managed, by means of an alphabetical table, to compose words expressing his thoughts.

A good many measures had been tried for arresting this paralysis. Faradization of the muscles of the tongue, soft palate, and lips, alone succeeded in temporarily restoring slight contractility to these enfeebled muscles, so that the patient was constantly begging for electricity to be used.

During the last month of his existence, deglutition became more and more difficult. As he could only use his left hand, a paste made with bread and wine, of semi-liquid consistency, had to be poured into his mouth. He first opened his mouth, letting his head fall backwards so as to receive the food, and then closing his mouth immediately with his left hand in order to keep the paste in, he bent his head forwards, making at the same time repeated efforts to swallow. In spite of this contrivance, it sometimes happened that the food came back through the mouth and nostrils. Subsequently, liquids alone could be swallowed, and the patient died at last of starvation fever, with rigidity of the limbs on the right side and paralysis of the bladder and rectum.

At the *post-mortem* examination, atrophy of the roots of the

hypoglossal nerve was found, together with increased consistency of the medulla oblongata.

At No. 19, in the same ward, we had an opportunity of studying another example of this form of paralysis.

B——, a gardener, aged 62, after having enjoyed excellent health previously, and having never committed any excess, or been exposed to any of those poisonous influences which sometimes bring on paralysis, fell ill in February, 1862. He was suddenly seized with fever and delirium, which lasted three or four days only. He was convalescent for a short time, and he seemed cured, when his attention was drawn by his friends to a slight nasal resonance of his voice, and he noticed himself that he had some difficulty in pronouncing words beginning with the letters *r, c, k, q,* so that the tongue was already somewhat embarrassed. The following month, at the end of a day's work in the sun, he suddenly felt weakness of the right leg and arm, without any impairment of intellect. He remarked at the same time also, that his food collected between his teeth and cheeks, and that at intervals he was obliged to wipe his lips, which were wet with the escaping saliva. His appetite was good, and all his functions were performed with regularity.

On June 12th, 1862, B—— was admitted into the Hôtel-Dieu, under Dr. Empis, who was then acting as Professor Rostan's substitute. He was still able to relate the accession and the course of his complaint, although his lips were manifestly paralyzed. He could not pronounce the letters *o* and *u,* and he dribbled when he talked. His face was natural when at rest; but when he laughed, the angles of his mouth were strongly pulled upwards and outwards, and his mouth remained half-open, so that he was obliged to use his hands in order to bring his lips together. The tongue seemed to be fixed behind the lower row of teeth, by which it was indented, and it was with great difficulty protruded outwards and forwards. Its apex, which deviated a little to the right, could not be raised to the upper incisors, or above the lower molars. The articulation of certain words, however, and deglutition were still possible, but with very manifest trouble and difficulty. The right arm and leg were weak; the left ankle could not be flexed, and the sensibility of the left side was diminished.

The progress of the disease was rapid and continuous. When, in September, B—— came under my care, he could no longer pronounce the letters *c, p, t;* but he could still articulate the consonants *b, d, l, m, n.* He swallowed his saliva with difficulty, and he already complained of a sense of constriction in the throat. He often passed his fingers down the back of his mouth, as if he wished to extract some foreign body which interfered with deglutition. His intellect was perfectly clear, and if he had

great difficulty in uttering sounds, his physiognomy showed that he understood perfectly all the questions that were put to him. Even then, however, the lower part of his face was not long before it became motionless, whilst the upper part, and more particularly the eyelids and forehead, retained all their mobility.

The feebleness of the sounds uttered by the patient was remarkable. Indeed, when his chest was exposed, one was struck with the weakness of his respiration. There was scarcely any oscillation of the walls of his chest during inspiration and expiration; the lungs took in and expelled very little air; expiration was feeble and slow. This was one of the reasons why the sounds were feeble. Besides, if he was asked to retain the air contained in his chest, he was unable to do so, and the air continued to escape slowly. The glottis remained always open, so that the air passed to and fro through the larynx, almost as through an inert tube. The glottis seemed to have lost the greater part of its active tension, and could no longer, under the control of the will, vibrate like strings, or like the membranes of a reed instrument. There was not only loss of speech, but complete aphonia also, and it was only by dint of considerable exertion that he could feebly utter the sound of *a*. The above details have already shown you how feebly respiration is carried on in such cases. In the man at No. 19 this difficulty of breathing was still more increased on his catching a cold. At such times he could not always cough, for he was not strong enough to expel rapidly the air contained within his chest, so that he could not easily clear his bronchial tubes and larynx of the accumulating mucus.

I was very much afraid lest the dyspnœa, which was very marked already, should go on increasing, in which case the patient would be choked through accumulation of the bronchial mucus. The muscles of the chest were therefore electrified every day, and, by his gestures, B—— expressed how much better he felt then. The dyspnœa became less intense, and for several hours afterwards the supplementary muscles of respiration, the sterno-mastoidei, the trapezii, and scaleni, ceased their rhythmical contractions, which had assisted the intercostal muscles and the diaphragm. Every day, however, until the cold got well, electricity had to be used.

The muscles of respiration were not the only ones affected, for the muscles of the neck were very feeble too. In fact, B—— could not touch his chest sharply with his chin, or keep his head forcibly extended. The cervical muscles, therefore, the trapezii, and sterno-mastoidei, shared in the weakness of the thoracic muscles, and perhaps also were the scaleni and the deep muscles of the anterior and posterior cervical regions similarly affected.

The patient had some difficulty in carrying his head up, and he had to pay a certain degree of attention in order to keep it in equilibrium.

Deglutition soon became still more difficult. The food, although perfectly masticated, passed with great difficulty, or with feeble jerks, from the cavity of the mouth into the pharynx. B—— then applied his hands over his mouth and cheeks in order to assist the contractions of the orbicularis oris and buccinators. The movement of elevation of the base of the tongue seemed very limited, and when the food reached the pharynx it was sometimes rejected through the nose. Liquids also were swallowed badly; they sometimes got into the larynx, in spite of the aryteno-epiglottidean folds, and brought on fits of coughing made up of short jerks.

The circulation, both centric and peripheral, presented no serious modification. The pulse at the wrist was a little more frequent than normal—92 in the minute; the heart's action was powerful and regular.

Until then, there was no paralysis of the bladder and rectum. In a short time, however, the general debility increased rapidly. The patient, who used to walk, although with difficulty, dragging his legs and resting on the back of a chair which he pushed before him, found himself incapable of leaving his bed. His breathing became slow and incomplete, deglutition more and more difficult, his facies altered, and death took place quietly and without any struggles, the patient having only a moment before made signs to thank the attendants for helping him to place his head down on his pillow.

Post-mortem examination.—No fatty degeneration of the diaphragm, although its fibres looked pale. No portion of the muscular system presented that beautiful red colour which is proper to it, and the extreme friability of the muscles of the right leg, especially of the peroneus longus, tibialis anticus, and quadriceps femoris, contrasted with the almost normal resistance of the corresponding muscles of the left side. Besides, the friable, softened muscles were of a reddish-yellow colour, and manifestly undergoing commencing fatty degeneration, a fact about which microscopical examination left no doubt.

The muscles of the face and the orbicularis oris, although not well developed, were not altered. The intrinsic and extrinsic muscles of the tongue were normal, as well as the buccinators, the muscles of the soft palate, of the pharynx, the larynx, and the neck.

The calvarium was very thin. The dura-mater looked thickened; the pia-mater was œdematous and injected, but could be removed without tearing away the cerebral tissue. The grey matter was of good consistency and unaltered; the white matter was of

a *café au lait* colour, and presented very distinct red points. Several portions of the circumference of the left corpus striatum were stained of an amber-red colour, which was apparently due to small hæmorrhagic clots of old date. These parts, when examined under the microscope with a power of 250 diameters, were seen to contain hæmatin in a state of fine powder, and granular deposits of a brownish-red tint.

The optic and olfactory nerves were of normal colour and consistency. The motor oculi had a greyish tint at its origin, but the fourth nerve was sound. The fifth and sixth nerves could not be examined at their origins. The facial was flattened at its origin, on both sides, but was not affected in other respects. The roots of the pneumogastric were atrophied, but the glosso-pharyngeal nerves were healthy. There was marked hyperæmia of the cerebellum. The floor of the fourth ventricle presented a plexiform arrangement of vessels.

The roots of the right hypoglossal nerve were so atrophied, that they resembled filaments of congested cellular tissue. When examined under the microscope, they were found to contain deposits of pink, brownish-red, and greenish hæmatin. The nerve-tubes were few in number, collapsed in parts, with a granular cylinder-axis, and apparently softening myeline. The roots of the left hypoglossal were not examined, because they had been torn from the bulb when the spinal cord was removed.

The roots of the spinal accessory were on both sides small. The neurilemma predominated, especially on the left side, and all the roots of the nerve, both those from the medulla and those from the spinal cord itself, were of a greyish colour. The microscope detected an increase of vessels in them; the capillaries of the neurilemma were turgescent, the neurilemma itself thickened, and consequently the nerve-tubes of the roots were distinguished with difficulty. In the midst of the elements of the neurilemma there was seen a fatty substance, irregularly scattered in granules. The fibres of the connective tissue were markedly developed, and were mixed up with a good many elastic fibres. The dura-mater for the upper third of the cervical portion was thickened, congested, and of an ashy-grey colour.

The anterior spinal roots were atrophied, especially on the left side, near the roots of the spinal accessory. In that part, the roots of the last nerve seemed to be reduced to a band of connective tissue, whilst the antero-lateral columns, in the part which gives attachment to the motor roots, were of the same colour, and had the same congested appearance as the posterior columns present in cases of locomotor ataxy.

The roots of the right spinal accessory nerve were less atrophied, but had to some extent the same colour, and were as congested as the roots of the opposite side. A good many of the anterior

spinal roots presented a relative diminution of size, and a markedly congested condition analogous to what has been noted in general progressive muscular atrophy.

Sections of the spinal cord, made at different parts, were examined, and marked hyperæmia of the upper cervical portion was found. The grey substance of the cord was of a deeper colour and was harder than natural, showing a relative sclerosis of the cord.

Are you not struck in this case, gentlemen, with the existence of general hyperæmia of the cerebro-spinal axis, coupled with relative atrophy of the greater number of cranial and spinal motor nerves? For these pathological lesions resemble those which have been described by Professor Cruveilhier in progressive muscular atrophy, and by Dr. Duménil (de Rouen) in a complex case of paralysis of the tongue and general muscular atrophy.

Let us now proceed to analyze Dr. Duménil's case, and the one reported in Dr. Duchenne's memoir.

Dr. Duménil reports his case under the following heading: *Atrophy of the hypoglossal, facial, and spinal accessory nerves: complete motor paralysis of the tongue, incomplete of the face. Integrity of the muscles of the tongue and face. Atrophy of the anterior spinal roots: incomplete paralysis of the limbs, incipient muscular atrophy.*—That atrophy of motor nerves should cause paralysis of the muscles to which they are distributed, is perfectly in accordance with physiological notions. But how is it that the atrophy of the spinal roots caused atrophic degeneration of the muscles supplied by them, whilst the atrophy of the cranial nerves and their roots did not produce the same effect on the muscles of the tongue and face? Dr. Duménil at first thought that this difference might be owing to the fact "that motor cranial nerves do not have the same influence on the nutrition of muscles as the anterior roots of spinal nerves." But Dr. Duchenne having reminded Dr. Duménil that in Professor Cruveilhier's case there had been noted atrophic degeneration of the tongue as well as atrophy of the hypoglossal nerve, Dr. Duménil had to give up his hypothesis, and, after fresh researches, he was perhaps the first to express the opinion that the impairment in the nutrition of the muscle was a consequence of a lesion of the sympathetic. However this may be, Dr. Duménil's patient was afflicted with a paralysis of the tongue, the muscles of the face and limbs, which was due to atrophy of motor, cranial, and spinal roots. How can an individual be said to suffer from two associated diseases, namely, progressive muscular atrophy and glosso-laryngeal paralysis, when the anatomical lesion is one and the same? We shall discuss this question by-and-by.

A case communicated by Dr. Costilhes to the Medical Society of Paris, in 1860, suggests the same reflections, since there was

general muscular debility as well as symptoms of glosso-laryngeal paralysis. However interesting that case may be, I shall now proceed to comment on the eighth case of Dr. Duchenne's memoir, which he calls: *Progressive paralysis of the tongue, the soft palate and lips, coinciding with progressive fatty muscular atrophy limited to a few muscles of the upper extremities.*

The patient stated that the disease had set in with weakness of the movements of the right arm. Dr. Duchenne found atrophy of the muscles of the right hand, and also commencing atrophy of the left hand, the trapezii, and many other muscles of the trunk and limbs. Whilst questioning the patient, he noticed besides a marked defect of articulation, a circumstance which surprised him at the *onset* of progressive muscular atrophy, because, in such cases, atrophy of the tongue is only observed at the *close* of the disease. Direct examination of the tongue, however, showed that the organ was not atrophied, but merely paralyzed, as were also the orbicularis oris and the muscles of the soft palate. The history of the case could not be completed, as the patient left off coming to Dr. Duchenne.

In this case, says Dr. Duchenne, there were two different diseases, namely, muscular atrophy of the limbs without paralysis, and paralysis of the tongue without atrophy. "Chance alone, a mere coincidence," adds the learned investigator, "had brought together these two distinct morbid varieties," both in the patient he saw himself in 1858, and in the one seen by Dr. Duménil in 1859.

I may be allowed to observe, however, that when Dr. Duchenne made this positive assertion, there was only on record the *post-mortem* examination made by Dr. Duménil, which established the existence of an identical lesion in the roots of the hypoglossal nerve and the anterior spinal roots. Since then, a *post-mortem* examination, made at my request by Dr. Luys and M. Dumontpallier, in presence of Dr. Duchenne himself, has shown that glosso-laryngeal paralysis and progressive muscular atrophy are attended with the same nerve-lesions, namely, atrophy of motor roots, both cranial and spinal. Besides clinical observation has proved to me, in all the cases which I have seen, that in patients suffering from glosso-laryngeal paralysis there is a tendency in the paralysis to become general. It is very probable, therefore, that there is more than a mere chance coincidence in all these cases.

I do not wish to insist further on this point, and I hasten to add that Dr. Duchenne was right in giving a distinct description of these two morbid conditions, because the progress of the disease is different, and the termination always rapidly fatal in glosso-laryngeal paralysis. But in my opinion these morbid states are only varieties of a paralysis depending on an affection

of the spinal cord or medulla oblongata, the *chief anatomical expression of which seems always to consist in an atrophy of the motor roots.*

Let us now return to the general study of the four cases of glosso-laryngeal paralysis which I have related to you. It is not difficult, if we keep in mind the principal symptoms observed in each of these cases, to give a broad sketch of this disease, the origin, progress, and termination of which are so very characteristic, that we meet with no other identical affection in the whole range of nosology.

When the patient comes to us for advice, the disease has already made great progress, and all its characters are well marked. On carefully questioning him, however, it is found that the first circumstance which attracted his notice was slight embarrassment of speech. Soon afterwards he observed that his tongue was not equally supple, and that his utterance became thicker and thicker. His food then lodged at times between his teeth and cheeks; the tip of his tongue being awkward and incapable of doing it, he had to use his fingers to replace the food on his tongue. The pronunciation of certain words was marked by a nasal resonance; the vowels *o* and *u* could not be pronounced because the contractility of the orbicular muscle, which is indispensable for this, had diminished, and there was occasional dribbling of saliva when the head was inclined.

Now do not these facts point to an incipient paralysis of the tongue, the soft palate, and orbicularis oris? By degrees, however, the paralysis makes continued progress; the tongue remains fixed as it were behind the lower teeth; its apex and its base are equally motionless: not a single word can be articulated. The first stage of deglutition has become almost completely impossible, and the patient has recourse to all kinds of stratagems for getting his food into the pharynx. He tries to help the orbicularis oris and the buccinator with his hands, and applying them over his mouth and cheeks, he makes repeated and considerable efforts in order to get his food to pass into the pharynx, and yet he takes great care to chew well what he eats, and to facilitate its gliding down by drinking and throwing his head backwards. At last he sometimes succeeds in swallowing, but at other times the co-ordinate contraction of the pharyngeal constrictors being at fault, only a small quantity of food gets into the œsophagus, whilst the greater portion is thrown up through the mouth and nostrils, the posterior apertures of the latter having remained open through the paralysis of the soft palate.

These unfortunate patients are of course a considerable time over their meals, for their appetite remains excellent. Liquids also are often swallowed with great difficulty. Oftentimes small portions of food pass into the larynx, and then to the horrible

torture of not being able to swallow is superadded extreme difficulty of coughing in order to get rid of the food which has passed into the larynx and trachea. The anxiety is extreme; at last, after frequent fits of a small jerking cough, the patient gets calm again. Hence it may be seen, that he is at every moment in imminent danger of death by suffocation.

When the paralysis has advanced so far, excessive weakness of the respiratory movements may be easily discovered. The walls of the chest scarcely move, and the diaphragm itself sometimes shares in this apparent immobility. At this period of the disease the auxiliary muscles of respiration have also become powerless, and superior thoracic breathing is impossible. If the patient be asked to blow out a candle, he collects all his strength, and yet the flame is scarcely agitated as he blows on it. This is not only owing to a division of the column of air which is expired, and its passing at the same time through the mouth and nostrils, nor merely to the inability of the patient to contract the buccinators and orbicularis oris in order to guide the column of air, but it is chiefly due to the small volume of this column, and to the paralysis of the bellows, namely, the walls of the chest.

If such patients be attacked with bronchitis, they are in danger of quickly dying of asphyxia, because they can no longer cough vigorously, and thus expectorate the bronchial mucus.

The pulse sometimes becomes frequent without any fever supervening, and I shall by-and-by inquire into the physiological reason of this frequency of the heart's action.

As a rule, no pain is complained of, but in some cases pain is felt in the occipital and upper part of the cervical region. Sensibility is everywhere normal: those very muscles which are paralyzed retain the property of contracting under the influence of electricity, and irritation of the mucous membrane of the soft palate produces contraction of the velum through a reflex action.

As the general debility makes constant progress, however, the patients drag themselves along with difficulty, resting on the arm or back of a chair, which they push slowly before them. They next refuse to get up, and prefer to sit up in bed, with the upper part of their body propped up, their head resting on pillows, and inclined to one side, in order to let the saliva which they are unable to swallow run out of their mouth. Their sleep is often disturbed by paroxysms of suffocation, probably due to the passage of the saliva or of the pharyngeal mucus into the larynx. If death does not take place in one of these paroxysms, it seems to be caused by an arrest of the contractions of the heart, is unaccompanied by pain or any noise, and occurs suddenly.

Such, gentlemen, is the course usually run by this malady, but it is sometimes accompanied by other morbid phenomena—by paralysis, for example, either of the upper and lower limbs, or of some muscles only of these different segments of the body. Such cases are merely instances of the extension of the disease. But in others you may observe genuine complications, such as atrophy and fatty degeneration of the muscles, hemiplegia even, due to there having occurred hæmorrhage or softening at a period anterior to the disease. In most cases, however, the patients die without presenting any other anatomical lesions besides those which are directly dependent on the disease itself.

Let us now inquire whether the alterations found after death can explain the symptoms observed at the bedside.

The first *post-mortem* examination which I made was entirely negative as regards the anatomical lesion; perhaps, however, because it was incomplete. In the second case there was found very marked atrophy of the roots of the hypoglossal nerve, without any alteration of the muscular fibres themselves. The medulla oblongata was apparently also of greater consistency than normal.

In the third case, I found well-marked thickening and grey discoloration of the dura-mater, on a level with the medulla oblongata, and as far down as the roots of the fourth cervical pair. This thickening was due to a considerable increase in the amount of fibres of connective and fibro-elastic tissue, and seemed to result from a chronic congestive process, as shown by the great number of capillaries and of deposits of hæmatin external to them.

The roots of the hypoglossal and spinal accessory nerves were atrophied, and reduced in several places to the neurilemma; and at the spot where the spinal accessory was in contact with the dura-mater there was adhesion of the neurilemma to the fibrous envelope of the cord, and a deposit of a nucleus of connective tissue of the size of a pea. A good many motor roots in the cervical region were thinner than natural, from partial disappearance of the nerve-tubes. With the aid of the microscope, the neurilemma was seen to preponderate everywhere over the nerve-tissue properly so called, and notable hyperæmia could be detected everywhere, also together with greyish discoloration of the neurilemma. The cord itself, at the upper part of the anterior columns, was as congested and of the same colour as the posterior columns are found to be in cases of progressive locomotor ataxy.

The fibres of the palsied muscles of the tongue, soft palate, lips, chin, and cheek, &c., were unaltered. As to the diminution in size of the muscles of the right leg, and their condition of incipient fatty degeneration, they need only be mentioned.

From the study of these three cases it follows, that in this complaint the paralysis is due to an alteration of the motor roots which supply the affected muscles, these latter in most cases presenting no change of volume and structure.

The complete paralysis of the tongue is accounted for by the general atrophy and complete disappearance, in some places, of the roots of the hypoglossal nerve. In Dr. Duménil's case, the alteration was not confined to the roots alone, for the trunk itself and all the branches of the nerve were of a greyish colour and notably atrophied. The lingual nerve, on the contrary, which is a branch of the sensory portion of the fifth cranial pair, was normal, as well as the glosso-pharyngeal, so that the healthy condition of these nerves accounted for the preservation of the general and special sensibility of the surface of the tongue. Electro-muscular sensibility had been present in the beginning, but had diminished by degrees, and the nervous influence which reached the muscle being feeble only, electricity gave little relief to the patients. Thus was explained, first the difficulty, and next the almost complete inability to swallow.

The embarrassment of speech and the modifications in the resonance of the voice are explained by the paralysis of the muscles of the tongue, soft palate, lips, and chin. Perhaps an alteration of the deep roots of the facial nerve would have been detected, had the examination been carried so far; but in default of this, Dr. Duménil found structural alterations of the trunk of the facial and its branches, which accounted for the loss of contractility of the orbicularis oris, which is indispensable for the pronunciation of the so-called labials, and particularly of the letters *o* and *u*. The alteration of the facial also accounted for the paralysis of the buccinators, the muscles of the soft palate and chin, which are supplied by this nerve.

We found, as you remember, grave lesions in the bulbous and spinal portions of the spinal accessory. Our attention was directed also to the pneumogastric, the roots of which were atrophied. The anatomical facts which we have mentioned account perfectly, therefore, for the principal phenomena which have been observed, and agree entirely with the results of physiological experiment.

The patient, however, had not only lost the faculty of speech, but had also become affected with nearly complete aphonia. This phenomenon depended on two causes, namely, paralysis of the muscles of the larynx and paralysis of the thoracic muscles. Indeed, physiological experiments prove that when the spinal accessory is torn off at its roots, aphonia is brought on through the relaxation of the vocal cords, which therefore become incapable of producing vocal sounds. On the other hand, Longet and Claude Bernard have shown that the section of both inferior laryngeal

nerves causes occlusion of the glottis during inspiration, and consequently death by asphyxia. This occlusion of the glottis can explain the sudden death of the patient, with this reservation, however, that in old persons as well as in old animals the complete occlusion of the glottis is not probable, on account of the considerable development of the anterior processes of the arytenoid cartilages, which leave between them an interval, open at all times, and called by Longet the *respiratory glottis*.

The absence of tension of the glottis explains the feebleness of the voice; whilst, through the spinal accessory being diseased, there can be no prolonged voluntary expiration, so as to sustain the voice, and when the patient, therefore, makes a great effort to utter a sound, he only succeeds in producing a short and low grunt.

In order to explain the feebleness of the voice, we must also keep in mind the weakness of the thoracic muscles, which scarcely inspire, and have consequently little to expire; and if at rare intervals a deep inspiration is made, perhaps it is to be ascribed to an affection of the pneumogastric. For is it not known that, whilst accelerating the heart's action, division of the vagus slackens respiration, and that the animal which is the subject of the experiment makes deeper inspirations at intervals? The feeble contraction of the diaphragm is explained by the lesion of the motor spinal roots from which the phrenic derives its nervous influence.

We thus see that physiological facts are in complete accordance with pathological observation to account for the symptoms or functional disturbances met with in this disease, namely, feebleness of the voice, slackening of the respiration, and death by suffocation or asphyxia. All these phenomena are the results of disease of the spinal accessory nerve.

The physiology of that nerve gives us also an explanation of other phenomena. "If to an animal in which the spinal accessory nerves have been torn off," says Claude Bernard, "appropriate food be thrown, it rushes on it voraciously; but it soon gets less ardent, and eating more slowly, stops and lifts up its head every time it swallows. If it be suddenly disturbed at that instant, a sort of cough or of sneezing is sometimes produced, as if portions of food had a tendency to pass into the trachea." Note, gentlemen, that the first stage of deglutition was normal in such cases, and that there had been no lesion of the hypoglossal nerve. This impediment in the second stage of deglutition is explained by the paralysis of the pharyngeal branch of the spinal accessory, but there is no complete paralysis of the pharynx, because its muscles receive other motor branches from the pharyngeal plexus.

Have we not, indeed, found in our cases that the food often

got into the larynx, and that the sensibility of this organ, which was unimpaired, then caused reflex contractions of its muscles, often, however, insufficient to expel the foreign body? These phenomena are analogous to those observed in animals after the spinal accessory nerves have been torn off, and in whose trachea and bronchial tubes, and upper lobe of the lungs even, portions of food may be found.

Sensibility, therefore, is preserved in the larynx as well as in the tongue and palate. The sensibility of the larynx, as you well know, depends on the superior laryngeal nerve, which supplies only one laryngeal muscle, the crico-thyroid. The use of this muscle is to swing the thyroid cartilage on the cricoid, and thus to tighten the glottis. The superior laryngeal is, therefore, partly a motor nerve, and, indeed, the experiments of Professor Claude Bernard have led him to believe that, although almost exclusively sensitive, the pneumogastric had still the power of exciting contractions. This motor power of the nerve is special, and might be termed *respiratory;* because, after the spinal accessory has been destroyed, and the functions of the larynx, as the organ of sound, abolished, respiration goes on when the animal is at rest; but if the pneumogastric be torn, or the recurrent laryngeal nerve be divided, the dilatation of the glottis is immediately replaced by a flaccid condition, and the animal dies of suffocation brought on by the approximation of the lips of the glottis during inspiration.

If the spinal accessory be an undoubted respiratory nerve, acting voluntarily on the muscles of the larynx and the supplementary muscles of respiration, the pneumogastric is an involuntary nerve, a nerve of organic life, which presides in the larynx as in the lungs over the maintenance of the respiratory functions. It is from the pneumogastric, then, that the laryngeal, tracheal, and bronchial mucous membrane, as well as the crico-thyroid muscles and the muscular fibres of the bronchial tubes, derive their sensory and motor properties; and this fact explains how oxygenation of the blood continues in cases of glosso-laryngeal paralysis, in spite of the lesion of the spinal accessory nerves and the anterior roots of the cervical and thoracic spinal nerves. That the vocal is not dependent on the respiratory larynx is again proved by comparative anatomy, for birds have a distinct vocal as well as a respiratory larynx. Lastly, is it not remarkable that in the complaint which we are now studying, the lesions at the onset are almost exclusively confined to the muscles of the life of relation, as shown by an alteration of the voice, of articulation, of expression, and physiognomy? While it is only secondarily that the tongue, the soft palate, and the pharynx, become affected as organs of deglutition and muscles of organic life. Later, however, and sometimes simultaneously

with, or even before the setting in of the impediment of speech, paralysis of some of the muscles of animal life is observed, as in the cases recorded by Drs. Duchenne and Duménil, and in the cases which fell under my own observation.

The healthy condition of the pneumogastric in some cases, and the slight degree in which it is affected in others, explain how it is that the other functions over which this nerve presides remain nearly normal. Thus, in no case was there paralysis of the œsophagus, or of the stomach; and secretion of the gastric juice, and gastric absorption, seemed to continue normally. As to the general debility and the wasting occurring during the last days of the patient's life, they are sufficiently accounted for, I believe, by the inability to swallow, by the patient's confinement to bed, and perhaps by the considerable loss of saliva through the opened mouth.

I have described to you, gentlemen, the chief symptoms of glosso-laryngeal paralysis, the *post-mortem* appearances met with, and I have attempted an explanation of the symptoms, that is to say, the pathological physiology of the disease, grounding my opinions on the anatomical lesions found, and on the learned experimental researches of physiologists. It now remains for me, in order to complete the description of this complaint, to draw your attention to its course, its modes of termination, and its differential diagnosis.

At the beginning of this conference I told you that glosso-laryngeal paralysis always terminated in death; and I do not believe that a single case of this disease is on record in which its progress has been arrested even for a few months. At the outset, however, the progress of the malady may be somewhat slow. The patient has an embarrassment in his speech for three, four, five, or six months, and he has some difficulty in keeping his saliva in his mouth; but as soon as deglutition becomes difficult, the disease makes rapid progress in most cases, and life is soon gravely compromised.

The disease, which had at first been apparently confined to the inferior segment of the face, and to the tongue, soon invades the larynx, the walls of the chest, and the diaphragm. The respiration, it is true, seems to be carried on with regularity still, but each inspiration is feeble, the patient seeming then to breathe after the manner of hybernating animals; and this incomplete respiration must, sooner or later, cause appreciable modifications in calorification and the oxygenation of the blood. In order to make the respiratory feebleness very apparent, it is only necessary to ask the patient to make some effort, when not only is feebleness observed, but also a want of harmony in the performance of the respiratory act.

The patient can no longer take in air enough to blow out a

candle; he can no longer keep up the amount of effort necessary to allow him to get into bed, or to walk a little briskly; still less can he go upstairs, for the least effort makes him pant for breath, and compels him to stop suddenly. He is unable to make an effort, because, owing to the paralysis of the spinal accessory, the aperture of the glottis remains wide open, and because the walls of the chest being no longer supported by the sterno-mastoid and trapezii muscles, which are now powerless, fall back on the lungs. From the inability of the inspiratory muscles to store up air within the lungs result the feebleness of the voice, and the disorders which must follow on deficient oxygenation of the blood, rendered sometimes still more imperfect by paralysis of the diaphragm.

Condemned to a nearly complete immobility, the patient is almost always in bed or sitting in a chair. For the same reason that he cannot walk, he cannot make the effort of coughing and expectorating, that is to say, he cannot make with his thoracic bellows the sudden inspiratory movements that are requisite for detaching the mucus contained in his bronchi in order to reject them by a violent expiration. This impairment of the chest-walls is a grave prognostic sign, because the least attack of bronchitis may, by causing engorgement of the lungs, kill the patient by asphyxia. Bronchitis, however, is not always a proximate cause of death, and indeed you saw that the patient at No. 19, in St. Agnes ward, did not die of the bronchitis which attacked him. We must admit, in such cases, that through a special organic contractility, the air-passages gradually rid themselves of their mucus by expelling it into the trachea and larynx. We almost have a proof of this hypothesis in the laryngeal embarrassment complained of by the patient in such cases. He is seen to make feeble efforts in his attempts to cough and clear his larynx; but he is unable to expectorate, and if the mucus be not immediately swallowed, it sojourns a variable time in the pharynx. In order to clear his pharynx, the patient again tries to cough, whilst, by passing his finger down to the back of his mouth, he produces a tendency to vomiting, through which the mucus is brought up as far as the base of his tongue, where he can seize it with his fingers.

In the description which I gave you of the disease, I did not lay great stress on the dribbling of saliva out of the mouth, a circumstance which is constantly observed, and which persists until the death of the patient. I did not speak to you either of the grave consequences which had been attributed to this prolonged loss of saliva, because there are on record cases of salivary fistula which, in man, and in horses also, did not bring on failure of strength or marked wasting. Dr. Vella, however, a professor at the University of Modena, and Dr. Duchenne, have

ascribed to this circumstance some share in the general debility observed in these cases.

But the progressive course of the paralysis of the muscles primarily attacked, and the implication of other portions of the muscular system, taken together with the pathological lesions observed, suffice to show the gravity of such a paralysis. The almost complete dysphagia, and the extreme frequency of attacks of choking caused by the passage of food into the larynx, give rise to fears that the amount of food taken will prove insufficient, and that death by asphyxia is ever imminent. Indeed, the patients die of starvation, and more frequently through being choked. Now when death is not preceded by any symptoms of pain or by spasms, has one a right to suppose that syncope was a proximate cause of the fatal termination? The patient at No. 19, in St. Agnes ward, probably died from a sudden arrest of the heart's action, and the *post-mortem* examination showed that the cavities of the heart were distended by large clots of blood.

There is another mode of fatal termination, by asphyxia, identical with that observed in cases of the general paralysis of the insane, and which is due to an arrest of the food on a level with the upper orifice of the œsophagus. This accident scarcely ever happens except at a period when the patient can still swallow semi-solid substances, whilst in the last stage of the disease he cannot accomplish this. From this observation may be deduced a therapeutic indication, namely, that life may still be prolonged for several days, or months, if the morsel of food be extracted in time. And, as you may recollect, the life of the woman lying at No. 29, in the St. Bernard ward, was thus prolonged.

Now, are we in a position, with the aid of the characters of this disease, to distinguish it from any other local or general paralysis?

The general paralysis of the insane sets in, it is true, with an embarrassment of the tongue; but there may be noticed, at the same time, slight convulsive trembling of the lips, and in most cases delirium is observed from the beginning, together with a fixed stare, which is never met with in the patients whose cases I related to you. Besides, in glosso-laryngeal paralysis the intellect is always perfectly clear, and the patients soon find out the gravity of their complaint; whereas this is not the case in the paralysis of the insane. Again, in this last affection, if sooner or later general feebleness of the muscular contractility be observed, in no case does this paralysis affect specially the muscles of the soft palate, nor is there ever dribbling of the saliva, whilst from the beginning the practitioner is led, on account of the failure of the intelligence, to locate the disease in the brain.

We need not stop to diagnose hemiplegia from this form of

paralysis, because, if in our patients we often found paralysis of one of the upper or lower limbs, we at the same time discovered disorders of motility in the muscles of the tongue, the soft palate, and the lips, which, taken as a whole, and from the symmetry of their manifestations, did not suggest the idea of a cerebral hemiplegia.

An affection, which is of very rare occurrence, namely, *double facial paralysis*, might be confounded with this form of disease, and the mistake would be excusable. Indeed, in double facial paralysis the muscles of the lips are motionless, and the patient has consequently a difficulty in pronouncing labials. On the other hand, if both facial nerves be diseased high up in the aqueductus Fallopii, the consequence will be that the patient's voice will have a nasal whine, owing to the paralysis of the soft palate. Let us add again that, through his inability to contract the isthmus faucium, he will have some difficulty in swallowing.

These symptoms resemble very much those of glosso-laryngeal paralysis, and yet these two diseases may be distinguished from one another. For in the former the hypoglossal nerve is not affected, and the tongue therefore is not impeded in its movements. In the latter, on the contrary, these movements are deeply interfered with. Again, in double facial paralysis all the muscles of the face are paralyzed, and whatever moral emotions be felt by the patient, his face preserves the immobility of marble. It seems, as Dr. Duchenne has felicitously expressed it, as if the patient laughed or cried from behind a mask. In glosso-laryngeal paralysis, on the contrary, the lower part of the face alone remains motionless, and if the patient laughs, he laughs with his eyes, and moves his zygomatici, and the muscles of his forehead. If he weeps, on the other hand, the upper part of his face is thrown into contraction and expresses true grief. In double facial paralysis, deglutition is scarcely affected, and it is only the articulation of the letters *o* and *u* which becomes difficult.

Glosso-laryngeal paralysis might possibly, in the beginning, when there is yet no great impairment of motility in the tongue and the orbicularis oris, be confounded with diphtheritic paralysis restricted to the soft palate, or implicating other muscles as well. But the fact of there having been a previous attack of diphtheritic angina, or a previous manifestation of diphtheria in some part of the organism, suggests the nature of the case, and the diagnosis will soon be confirmed by the isolated localization of the paralysis in the soft palate, or, in cases when it becomes general, by other functional disorders which are never observed in glosso-laryngeal paralysis, namely, modifications of the general sensibility and special disorders of vision.

In the cases when progressive muscular atrophy begins in the tongue, and next attacks the soft palate and the orbicularis oris

simultaneously, or posteriorly affecting the muscles of the limbs and trunk, a mistake might be made. Progressive muscular atrophy, rarely, however, begins in that way in the adult; and even were it to do so, a careful examination would soon disclose well-marked muscular atrophy of some other part of the body, in most cases in the thenar and hypothenar eminences, the interossei muscles of the hand, &c. Besides—and Dr. Duchenne lays great stress on this fact—in glosso-laryngeal paralysis the paralysis sets in at once, unaccompanied by atrophy; whilst in progressive muscular atrophy, the atrophy is primary, and paralysis supervenes only after the destruction of the contractile fibres.

There are on record some very interesting cases, which Dr. Duchenne has termed cases of associated diseases, in which progressive fatty muscular atrophy affecting the limbs is met with concurrently with paralysis, without atrophy, of the muscles of the tongue, the soft palate, and the lips.[1] Dr. Duchenne thinks that there are two distinct diseases associated in such cases. But must we entirely concur in this opinion? When in the same individual you find, on the one hand, progressive paralysis of the tongue without any atrophy of the organ, and, on the other hand, progressive muscular atrophy in other parts of the body, will you not incline to the opinion that these two morbid conditions are dependent on the same organic lesion? Lastly, if pathological anatomy proves to you that the roots of the hypoglossal and the spinal motor roots have undergone the same alterations, can you refuse to believe that the *same* anatomical nerve-lesion has produced in one part paralysis of the tongue without atrophy, and in another part, paralysis with fatty degeneration of certain muscles?

Dissection has shown that, in glosso-laryngeal paralysis, the lesion was primarily seated in the upper portion of the cord and in the motor roots. We saw that, in that part only, had the dura-mater acquired considerable thickness, and that it presented a highly vascular condition, with greyish discoloration, pointing to congestion of ancient date. We saw the roots of the spinal accessory nerve reduced to their neurilemma, and we noted incipient atrophy of the cervical roots.

These anatomical details sufficiently indicate the gravity of glosso-laryngeal paralysis. But does it follow that, in no case, is the physician able to help the patient? It is plain, that in the two first stages of the disease the physician can, I do not say arrest completely the progress of the disease, but at least prevent it from being so very rapid, and can relieve for some time. He is still able, with only one remedy, namely, Faradiza-

[1] *Vide* Case VIII. of Dr. Duchenne's memoir, and Dr. Duménil's case, *Gaz. hebd.* 1859 and 1861.

tion of the affected muscles, to restore to them a transient contractility, and thus obtain that deglutition be accomplished with a little less difficulty and pain, and consequently that food be taken more regularly and effectually. He may, by galvanizing the auxiliary muscles of respiration, the intercostal muscles, and the phrenic nerve, favour the action of the contractile agents of thoracic and diaphragmatic breathing. But the power of the physician does not go beyond this, and little trust is to be placed on the passing of probangs down the œsophagus, and on the administration of strychnine.

Lastly, does the nature of the disease point to a special method of treatment? Nothing has been or could be tried on that ground, since those who studied the complaint having only the interpretation of the symptoms to guide them, could only conclude in the existence of a paralysis of undetermined causation. An injury or the rheumatic diathesis could not be invoked as the cause; nor could any poison in the blood account for the phenomena observed. So that the symptom paralysis could alone be combated. Let me add that the seat of the first manifestations of the disease, and the absence of all cerebral symptom, did not admit of the supposition that the morbid cause was seated in the brain. The pain in the occipital and cervical region, as well as the sensation of pharyngeal constriction, could only suggest the idea of an inflammatory lesion of the bulb and the upper portion of the cord, in the same way as the functional disorders led one to believe that the hypoglossal, the spinal accessory, and the spinal nerves were perhaps diseased at their roots or in some point of their course. But the occipital and cervical pain was not present in all the cases. And even if a lesion of the nervous system could have been almost affirmed, the hypothesis of an anatomical lesion could have been expressed with some reservation only.

Dissection alone could shed light on this twofold question of morbid nature and etiology. The *post-mortem* appearances found in the patient No. 19, St. Agnes ward, pointed to extensive lesions, which, together with those which we had already found in the patient No. 23 in St. Agnes ward, and the distinct statements made by Dr. Duménil, constitute together an amount of information of considerable importance.

From all these facts it follows that, in glosso-laryngeal paralysis, anatomical lesions may be met with characterized by the atrophy of the roots of motor nerves, namely, the hypoglossal, spinal accessory, and spinal nerves. This atrophy, which is thoroughly identical with that described by Professor Cruveilhier and other observers, in cases of progressive muscular atrophy, seems to be the result of a congestion of ancient date, causing the gradual disappearance of the nerve-tube, and hypergenesis of the connective tissue and neurilemma of the motor roots.

The spinal cord itself participates also in the same congestive process.

It now remains to determine whether this hyperæmia is of an inflammatory nature; and if inflammation be once admitted, the predisposing and exciting causes of this inflammatory process will have to be investigated, and the point determined whether it does not depend on a special diathesis.

To try and solve such problems would be opening up a vast field to hypothesis. We are at present in possession of no fact which authorizes us to discuss any of them. I prefer taking only into account the hyperæmia, as shown by an exaggerated vascular condition, the deposits of hæmatin, and the hyperformation of the connective tissue. We should, therefore, merely seek for remedial measures capable of combating this hyperæmia. And even then we can hope to interfere with some degree of success only at the outset of the disease, in the stage of congestion; for when the anatomical alteration has been once produced, no practitioner could ever think of making fresh nerve-tubes and of regenerating a portion of the spinal cord.[1]

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[1] [The following case, which came under my observation at the National Hospital for Paralysis and Epilepsy, is an instance of the rare combination of progressive paralysis of the tongue, soft palate and lips, with ordinary progressive atrophy affecting the muscles of the limbs.

Timothy M——, aged 45, a labourer, married, residing at West Haddon, Northamptonshire, was admitted on November 15, 1864. According to his wife's statement, his previous health had been excellent, and he had been a strong, stout, and hearty man; but he was then considerably reduced in size, and remarkably weak. His illness only dated eighteen months back, and he could not trace its origin to any cause except perhaps constant exposure to wet, as he had for some time previously been engaged in draining a field. The first symptom which attracted his attention was weakness and numbness of the right upper limb, which were soon followed by thinning of the hand and the rest of the limb, as well as by gradual loss of power, increasing in proportion as the wasting grew more marked. Six months afterwards he began to complain of a dull aching pain, exactly localized in the lower part of the back of his head, whilst his right leg felt weak. About fifteen months after the manifestation of the first symptoms of illness, his wife noticed that his speech became thick and indistinct, and that within a short time this embarrassment had become so marked that he was unable to articulate certain words. He at the same time began to complain of some difficulty in swallowing. Throughout his illness his intellect had been unaffected, and his memory had not failed. His sight and hearing had remained as good as ever. His appetite had been always good, and yet he was losing flesh every day. Since the difficulty of swallowing had set in, his complaint had made considerable progress; the weakness of his right leg had increased, so much so that he had of late been scarcely able to move about, even with the help of a stick, whilst his right arm had become completely useless to him.

When I first saw him, in November 1864, I could not make out what he attempted to say to me. He could not articulate a single word, and, in fact, the only sound which he could utter distinctly was that of the letter *a*. When he wanted to say *no*, he could only succeed in bringing out a sound like that of

haw. The orbicularis oris was paralyzed, so that he could not approximate his lips, and through his constantly half-opened mouth his tongue could be seen, shrunk and corrugated, lying flat on the floor of the cavity behind the lower row of teeth. There it lay perfectly motionless, and no effort of his could succeed in protruding or moving it in the least. Its sensibility to touch and pain was perfect, however, and the patient indicated by his gestures that he could taste as well as before his illness. On asking him to open his mouth wide, and to draw in his breath, the soft palate was seen to remain motionless, instead of rising as it normally does. The mouth was full of a thick, viscid, somewhat ropy saliva, which kept dribbling out whenever the head was slightly inclined forwards. The voice, besides its nasal resonance, which was due to the paralysis of the soft palate, was hoarse, and although the patient made considerable efforts to speak out loud, it continued feeble and low, partaking somewhat of the character of a grunt. Deglutition was gravely impaired; solids could not be swallowed at all, and liquids alone, or pastes of semi-liquid consistency, could with difficulty be got down. The patient had to be fed, and when his food was of the consistency of thick paste, he pushed it down his throat with the fingers of his left hand. Very frequently he got choked whilst eating or drinking.

The right arm was bent at the elbow, somewhat stiff, and completely useless. The muscles of the hand and of the fore-arm were entirely gone, those of the arm less so, and were seen to quiver when exposed. The hand had the characteristic aspect of what Duchenne has called the "*main en griffe* (the bird's claw hand). The right pectorales and deltoid were of diminished bulk and consistency, and were also the seats of well-marked fibrillary action. The fleshy masses of the right gluteal region of the thigh and leg on the same side were wasted, and, when slightly tapped with the finger, became affected with the fibrillary quivering which is characteristic of progressive muscular atrophy. All these parts were also subjectively as well as objectively cold.

On the left half of the body, the hand alone seemed to be affected, and only partially so, the atrophy being confined to the inter-ossei and lumbricales muscles (as shown by the deepening of the inter-metacarpal spaces), and to the muscles of the ball of the thumb. The hypothenar eminence was normal. The bladder and rectum were unaffected.

The patient remained under observation in the hospital for nearly five months. Treatment had not the slightest effect in modifying or arresting the disease. The muscles of the neck, which, on admission, were not markedly affected, became gradually atrophied; the sterno-mastoidei and trapezii, on both sides, diminished in size and grew soft and flabby, whilst the deep muscles of the neck themselves were probably diseased also, for the patient could not balance his head and keep it erect, and it would either drop down on his chest, or, if not supported by pillows, would fall backwards. The pain at the lower part of the occipital region and the top of the nucha was constantly complained of; it was only partially and temporarily relieved by flying blisters. The difficulty in swallowing grew daily worse and worse; sponge-cakes well soaked in milk and meat chopped very fine were at first taken in small quantities, and with great difficulty swallowed; but after a time nothing but liquids and eggs beaten up with wine could be got down. Sometimes some of the food passed into the larynx, and provoked awful spasms and a sense of suffocation, making the patient look as if on the point of death. On several occasions, the gasping for breath was followed by fainting, which nearly proved fatal. These paroxysms of intense dyspnœa sometimes came on independently of any food or drink getting into the larynx. Respiration was very feebly carried on, and it seemed to be in part kept up by an exercise of the will, for the patient could not sleep for more than an hour or two at a time, and often started up from sleep, feeling suffocated.

In the beginning of January, 1865, he could scarcely bear the exertion of getting out of bed, and kept his bed almost constantly, propped up by pillows in a semi-sitting posture. His appetite could never be satisfied, and he was always craving for food, pointing to his epigastrium with tears in his eyes. He had

several attacks of diarrhœa, which were easily arrested, but which always left him weaker than before. At the end of March he insisted on leaving the hospital, and returning to Northamptonshire. He there lived on new milk, and died recently only at the end of June. During the last week of his existence he could not manage to swallow the milk, and he gradually sank without struggle or pain.

It is very much to be regretted that a *post-mortem* examination could not be made in this case. The symptoms observed during life were in every respect so similar to those described by Professor Trousseau in the above Lecture, that there is no doubt but that the same anatomical lesions would have been found as in his cases. Dr. Duchenne (in "Traité de l'Électrisation localisée," p. 644) states that the tongue is very rarely implicated in cases of progressive muscular atrophy, and that he has met with this complication in 13 only out of 159 instances of the disease collected by him. He lays great stress on the fact that, in such cases, the tongue is never completely paralyzed at the beginning, but merely weakened in its movements; and he points to this as a diagnostic sign helping to distinguish this complication of Cruveilhier's disease from the progressive paralysis of the tongue, soft palate, and lips. In the case of T. M——, the paralysis of the tongue was complete, and I saw him only three months after the embarrassment of speech had set in. This paralysis of the organ, moreover, co-existed with characteristic atrophy of the muscles of the right half of the body and of the left hand.

Now, in such a case, were there two different and distinct diseases, occurring simultaneously, through a mere coincidence, in the same individual? Or was there only one disease, depending on anatomical lesions of the same nature? The absence of a *post-mortem* examination precludes the possibility of giving a positive answer to this question. But reasoning from analogy, and keeping in mind the cases observed by Dr. Duménil and Professor Trousseau, there can scarcely be any doubt as to there having been only one disease here present, depending on one and the same kind of anatomical lesion—namely, atrophy of a certain number of motor roots.—Ed.]

LECTURE VI.

PROGRESSIVE LOCOMOTOR ATAXY.

(PROGRESSIVE LOCOMOTOR ASYNERGIA.)[1]

§ 1. Definition.—Prodromata: Pain, Disorders of Innervation.—Nocturnal Incontinence of Urine; Spermatorrhœa.—Paralysis of the Third and Sixth Cranial Pair.—Diplopia.—Amaurosis.—Symptoms: Defect of Co-ordination of Movement with Retention of Muscular Power.—Transient, Persistent Pain.—Impotence.—Deafness.—Varieties: Painful Ataxy; Ataxy more marked on one Side of the Body.—Etiology: Hereditary Influence.—Symptoms of the fully-developed Disease.—Disorders of Progression.—Spasms.—Variable Anæsthesia, which is sometimes completely absent.—Return of the Paralytic Symptoms.—Progressive Locomotor Ataxy may be imperfectly developed.—Course of the Disease.—Prognosis extremely grave.—Locomotor Ataxy independently of Cutaneous and Muscular Anæsthesia.—A Few Words respecting Sir Charles Bell's *Muscular Sense* and Gerdy's *Sense of Muscular Activity*.—Differential Diagnosis between Progressive Locomotor Ataxy, Various Forms of Paralysis, and Cerebellar Ataxy.

Gentlemen,—You have had occasion to see several cases of *progressive locomotor ataxy* in my wards, and I have repeatedly called your attention to them. In 1861 and 1862 I devoted several lectures to the study of this singular malady, and I return to-day to the subject because recent discussions have imparted fresh interest to it. Formerly the pathological anatomy of the disease was very incompletely known, but cases that have occurred in my own practice, and others recorded by physicians of note, have since supplied us with interesting facts which I desire to bring before you. First, however, allow me to give Dr. Duchenne (de Boulogne) the credit which is due to him, and which has lately been contested by some. There is nothing surprising indeed that, before Dr. Duchenne wrote on *progressive locomotor ataxy*, cases, evidently referable to this disease, should have been seen and recorded by others. Locomotor ataxy is not a new disease, and Dr. Duchenne has never pretended that he was the first to suspect its existence. Such cases, however, had not been

[1] [The word *asynergia* would be better than that of *ataxy*, which has already a definite sense in medical language, different from its meaning in locomotor ataxy; but as this latter term has been almost universally adopted in France, I have hesitated before changing it.—*Tr.*]

seen in their true light, and the few descriptions of the disease given by foreign authors, under different and more or less appropriate names, were, to say the least, very incomplete. I do not even except that of Professor Romberg (of Berlin), whose monograph on the subject has, however, been called a masterpiece of conciseness and exactness. I admit that it is concise, but I deny that it is exact, both as regards the description of the symptoms and the pathological anatomy. I make this assertion after a careful perusal of the translation, which Dr. Zubelsky (of Varsovia) kindly wrote for me, of the chapter on Tabes dorsalis, in the edition of 1851 of Romberg's work.[1]

Even admitting, for the sake of argument, that the researches of German and English physicians on this subject be as complete as they are stated by some to be, it must be acknowledged that in France, as well as in England and Germany, the attention of the profession has been drawn to this subject only since Dr. Duchenne (de Boulogne) published his memoir.[2] It is to him, therefore, that we are really indebted for the knowledge we now possess of an affection which, until then, had been confounded and mixed up with such very different diseases.

As to the name, *progressive locomotor ataxy*, given to the complaint by Dr. Duchenne, I accept it, however long it may be, because it conveys to the mind, I believe, the most complete idea of the disorders of locomotion which constitute the most striking phenomena of the disease. The terms *atrophy of the posterior columns of the cord*, and *tabes dorsalis*, which have been suggested in its stead, are not better, in my opinion. The name of *tabes dorsalis* has only its antiquity to recommend it, and it has the disadvantage of having been applied by the ancients, and by others since, to very various affections, especially, as in the works of Hippocrates, to special paralysis brought on by sexual excess.[3]

I likewise reject the denomination of *atrophy of the posterior columns of the cord*, first, because it is as long as the one which I adopt, and secondly, because it is not so precise as some would have us believe. For, as I shall have to tell you when we come to the pathological anatomy of the disease, cases have been recorded in which the distinctive characters of progressive locomotor ataxy have been present, and that during several years, whilst after death no material alteration of the posterior columns of the cord has been found.

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[1] [See "Romberg on Diseases of the Nervous System," translated by Dr. Sieveking, vol. ii. pp. 395—401.—Ed.]

[2] "De l'Ataxie locomotrice progressive (*Archives Générales de Médecine*, Décembre 1858, Janvier, Février, et Mars 1859), and "le Traité de l'Electrisation localisée," by Duchenne (de Boulogne), 2e édition, Paris, 1861, pp. 547—620.

[3] Consult on this point, chap. xiv. De Internis Affectionibus, and chap. xix. of lib. ii., De Morbis, in the works of Hippocrates.

Now, gentlemen, what is meant by *progressive locomotor ataxy?* According to Dr. Duchenne (de Boulogne), the fundamental characters of the disease are—"Progressive abolition of the faculty of co-ordinating movements, and apparent paralysis contrasting with the integrity of the muscular power."[1] This is a very incomplete definition, however; but, for the present, I shall not attempt to give you another myself, for definitions in general —and in medicine perhaps more than in any other science— are not easily framed. They become still more difficult, nay impossible even, when they must be applied to a recently known disease, or, at least, a disease which has been but recently studied, and presenting an infinite variety in its manifestations, and the order of their sequence.

If you ask an individual suffering from ataxy to walk, he staggers, makes great efforts to maintain his equilibrium, and, feeling that his muscles do not respond to the influence of his will, he seeks for a point of support. It is especially at starting that this difficulty in maintaining the equilibrium of the body is remarkable. When once started, the patient is able to walk, although he does it badly, and throws his legs about to the right and to the left. Occasionally he loses his equilibrium entirely and falls down, unless he be supported, especially when he turns round. Formerly, a man whose gait was uncertain, whose legs were thrown to the right and to the left, was set down as suffering from paralysis, and if no serious impairment of the intellect were present, the disease was localized in the cord, and called paraplegia. No physician, before Dr. Duchenne (de Boulogne), ever thought of testing the muscular power of these so-called paralytic patients. The idea first occurred to this *savant*, and he it was who detected that their muscular power was considerable, and that they only lacked the faculty of co-ordinating their movements. You have yourselves examined my patients in St. Agnes ward who are suffering from locomotor ataxy. The one, in bed No. 2, is a young man whose muscular power is so great that his limbs cannot be flexed or stretched against his will. Although his gait be so vacillating, he is strong enough to bear on his shoulders, when standing, a weight of 160 lbs., on condition, however, that he may rest on a friend's arm, or on a piece of furniture; and I showed you that he could carry on his shoulders several students in succession. Surely this is not muscular weakness, and still less paralysis.

At No. 23, the patient was about 40 years old. He, too, looked as if he were paralyzed, for his gait was tottery, the least

[1] "De l'Électrisation localisée et de son Application à la Pathologie et à la Thérapeutique." 2e édition, Paris, 1861, p. 547.

touch sufficed to throw him down, and he could not walk across the ward, except by going from bed to bed. When sitting or lying down, however, his limbs could not be extended or flexed against his will.

Look now at that woman in bed No. 23, St. Bernard ward, and at that man lying at No. 11, St. Agnes ward. Both of them possess considerable muscular power, yet when they are up, even though they be propped up under the arms, they cannot move a single step, they thrust their legs forwards, backwards, and laterally, in a strange disorderly manner. When their eyes are closed, this disorder knows no bounds; their movements become so extravagant that they baffle description, as you saw yourselves. If the strength of their muscles be tested, however, whilst they are in a sitting or a lying posture, one is surprised to find it unimpaired, or nearly so, and to find also that unless considerable efforts be made, the limbs of these so-called paralytics cannot be flexed or extended against their will.

The difficulty which these patients have in co-ordinating their movements, is still more marked when they have not the sense of sight to guide them. But it must be observed, however, that the sight can never completely remedy the want of co-ordination in ataxy, whilst this obtains in cases of the mere loss of tactile sensibility, as we shall more fully state when treating of the differential diagnosis. The difficulty which the patients have in guiding their movements is much more marked when they first start, and when they turn round. It diminishes when they can rest on something, especially on a friend's arm. In some rare instances, the disease is restricted to this defect in the power of co-ordinating voluntary movements, and is unaccompanied by impairment of muscular sensibility, by analgesia, or by cutaneous anæsthesia. In other words, all the functions of the cerebro-spinal system are performed normally, with the exception of the faculty of co-ordination.

Note, however, that this form is very rare, I may even say, exceptional. Since my attention has been drawn to locomotor ataxy, I have seen more than fifty cases of the disease, and in three only have I seen it consist merely in a want of co-ordination. One of these cases occurred in a gentleman eighty years old, residing at Tours, and a patient of Dr. Duclos. He was suffering from paraplegia, and as his case seemed to his medical attendant to differ from one of ordinary paraplegia, I was consulted. The patient looked in excellent health, although he had not for a long time been able to walk. He generally sat up in a chair, and for the last twelvemonth he had had some paralysis of the bladder. A few days previous to my visit, Dr. Duclos had been struck with the extraordinary suddenness and violence with which the patient had stretched out his leg, when asked to do

so. On my testing, in my turn, his muscular power, I could not succeed in flexing or extending his legs against his will. I then made him get up on his feet, and by letting him rest on my arm he was able to carry on his shoulders his own medical attendant who had pronounced him to be paralyzed.

The mistake, however, was very excusable, and every one made it a few years ago. Even one of the most distinguished professors of the faculty, a man of very extensive knowledge, was deceived himself in the case of a patient whom we saw together at Tivoli. But he was easily convinced, however, that there existed no muscular paralysis, when there was only a want of co-ordination. This case of the old gentleman was one of simple ataxy, and as to the slight paralysis of the bladder, it could be ascribed to his advanced age. In 1860, however, I was asked by my excellent friend Dr. Deguise to see with him a superior cavalry officer, who was very markedly ataxic. The sensibility of the skin, the muscles, and joints had undergone no modification. There was nothing wrong with the eyes, the bladder, or intestines. The case being clearly one of uncomplicated ataxy, I showed it to Dr. Duchenne.

Now, how does the disease begin ?

Its accession is marked by various neuroses, and one of its premonitory symptoms is *pain*. How many patients, that have been sent to the baths at Néris, Bourbon-Lancy, Bourbon-l'Archambault, and Bourbonne, for rheumatic or pretended neuralgic pains, and who derived no benefit from the baths, were perhaps suffering from the pains which usher in locomotor ataxy? The characters of these pains are peculiar : they come on and go off with the rapidity of lightning or of the electric spark ; in some cases, however, lasting from a few seconds to a minute. They recur ten, fifteen, twenty times in an hour, and they come on in paroxysms several times in the year, or in a month, often without any other exciting cause than variations of temperature. At other times they are of a boring character, and either simultaneously or successively attack limited, perfectly well-defined spots, which the patient quickly compresses or rubs so as to diminish the pains. When the disease is confirmed, as I shall tell you by-and-by, these pains may become continuous and gradually increase in intensity. These have been described by some authors under the names of *general neuralgia* and *neuralgic rheumatism*, but Dr. Duchenne was the first to point them out as the prelude of locomotor ataxy. They are the most constant premonitory symptom of the disease, and yet in September, 1861, I had under my care at the Hôtel-Dieu a man aged 37, suffering from well-marked ataxy, who had never had any pain.

Nocturnal incontinence of urine is another neurosis which may precede locomotor ataxy. More frequently, and in nearly half the

cases which have come under my observation, there had been spermatorrhœa. The seminal losses were either diurnal or nocturnal. In the former case they occurred chiefly during defecation, from compression of the vesiculæ seminales. In Lallemand's work on spermatorrhœa, you will find several cases of paraplegia which were certainly cases of locomotor ataxy. At No. 23, St. Agnes ward, was a patient of mine who had for years been subject to spermatorrhœa, which had exhausted him considerably. Frequently these nocturnal emissions are accompanied by erection and voluptuous sensations; but in some cases there is anaphrodisia instead of spermatorrhœa, marked by an imperfect erection or a complete absence of sexual appetite.

There is again another form of genital neurosis in ataxic patients; namely, a singular aptitude for repeating the venereal act a great many times within a short period. This is an abnormal condition in man; for if birds, and some mammalia, such as the ram, the bull, and deer, can have connection rapidly, and repeat the act at short intervals, in man the act must extend over a certain time, and if performed too quickly it indicates a deviation from health. Men who possess this semblance of exaggerated virile power are often subject to spermatorrhœa. Only yesterday you heard the patient lying in bed No. 2, St. Agnes ward, confess that before his admission into the hospital he was able to have connection as many as eight and nine times in one night. Recently again I saw in my consulting-room a gentleman in the prime of life, and suffering from ataxy, who told me that he could have connection eight or ten times in the twenty-four hours. That this condition is abnormal is proved by there having most frequently existed incontinence of urine at some previous period, and that involuntary seminal emissions often occur.

Certain forms of transient paralysis also precede the want of co-ordination. I was lately consulted by a gentleman from the Côte-d'Or, who nine months ago was suddenly seized with left hemiplegia. There was no impairment of the intellect, and he could resume his occupation at the end of a week. The hemiplegia could not have been due to cerebral hæmorrhage or softening, nor was it probable that it could have been caused by cerebral congestion, since there had been no loss of consciousness, not even temporarily. Paralysis of the fifth cranial pair, which had occurred simultaneously with the hemiplegia, persisted, and in July of the same year the patient was seized, on two different occasions, with paralysis of the tongue, of a few seconds' duration only. From that time, however, his gait became uncertain, and the locomotor ataxy soon made frightfully rapid progress.

These instances of transitory paralysis are rare. The paralysis very often lasts some time, as when it affects the sixth cranial

nerve, producing on a sudden internal strabismus, or when it attacks the third or motor oculi nerve, causing external strabismus, diplopia, and ptosis. The duration of these forms of paralysis is very variable; they may last for the remainder of the patient's life, or they may go off after a few months, or even after a few days only. In some cases they recur, when the disease is fully developed, after having disappeared for several years.

This is the form of paralysis which, as it gets well spontaneously, has made the fortune of so many methods of treatment, whilst the very success of the treatment contributes to leave the medical man in error. Paralysis of the third and sixth pair has been looked upon by many pathologists as dependent on constitutional syphilis, and when a treatment by mercury and iodine has been followed by apparent success, the diagnosis seems to be confirmed, and the other phenomena which characterize ataxy are ascribed to the same cause. Within a short time, however, the same remedies prove utterly powerless. Vision itself may be deeply affected. Amblyopia, for instance, may be present for some time; or the patient may have amaurosis on one side, and discover it by chance; or the amaurosis may be double, as in the case of the man in bed 23, St. Agnes ward.

On carefully examining ataxic patients in the intervals when they are free from pain, there is often noticed an injection of the conjunctiva, sometimes as marked as in the most violent conjunctivitis, and in some cases giving rise to a sort of chemosis. There is at the same time contraction of the pupil, reducing it to the smallest possible size, and so powerful sometimes that it resists the influence of belladonna. On the other hand, during the paroxysm of pain, especially when the pain affects the head, the contraction of the pupil is replaced by more or less marked dilatation, and generally also the vascular injection of the conjunctiva disappears at such times. I merely mention these facts now, but further on I will revert to them, and try to interpret them.

Other cranial nerves may be affected as well, although this is the exception, and not the rule. These affections may coincide or alternate with those I have previously mentioned. Thus, the auditory nerve has been found paralyzed either on one or both sides, and I shall give you an instance of this presently. Dr. Duchenne has twice met with paralysis of the fifth pair concurrently with that of the third. "In one of those cases the two nerves were affected on the same side; in the other the fifth was paralyzed on both sides, and the third on the left only: in this case there was also paralysis of the soft palate and larynx."

Some of these premonitory nerve affections may be absent, but it very rarely occurs that they are all absent in the same case. I have nearly always found them, and Dr. Duchenne is

right in attaching great importance to them for diagnosing the disease at the outset. Remember, besides, that they may have been transitory, and been forgotten by the patient, so that the physician must needs make careful inquiries in order to discover their existence in the patient's previous history.

The accession of the disease is again marked by strange sensations, by a sense of constriction of different parts of the body. The patient feels as if his chest, his arms, or legs, were compressed by an india-rubber cuirass. His shoes feel too tight, and he often has the sensation of a belt constricting his abdomen. And just as in the most confirmed cases of paraplegia, there is paresis of the rectum and bladder, or even paralysis of their sphincters.

The etiology of the disease is still very obscure, and Dr. Duchenne and I have not been able to discover constant causes in the cases which have come under our observation. The cases on record are, however, sufficiently numerous now to admit of my making a few remarks on the influence which age, sex, and hereditary predisposition seem to have on the production of the disease.

Locomotor ataxy is chiefly met with about the middle period of life, from 20 to 40, although it may occur late in life, as in the case of the gentleman 80 years old, which I related to you. It is a remarkable fact that males are more prone to it, and that in a very large proportion. Dr. Duchenne has only seen it four times, and I three times, in females.[1] The general paralysis of the insane is another affection which greatly preponderates in the male sex.

Now, what influence has hereditary predisposition on locomotor ataxy? If this question can be answered, with great difficulty only, in the case of progressive muscular atrophy, the difficulty is greater still in the case of locomotor ataxy, which has been but recently studied. If you find, however, in the patient's family history, that there have been cases of various nervous diseases, you will be in a certain degree authorized in connecting ataxy with those diseases, and ascribing to them a common origin.

When treating of epilepsy, as you may remember, I related to you the history of a family, the different members of which were afflicted with monomania, hypochondriasis, epilepsy, seminal losses, and locomotor ataxy—thus illustrating what I told you of the transformation of neuroses into one another.

On July the 17th, 1861, a physician of Rouen brought me a patient, aged 45, who was suffering from locomotor ataxy, in a very advanced stage. His intellect was perfect, but an uncle and an aunt of his were insane, one of his brothers was ataxic and another and younger brother was hemiplegic.

[1] [One of the cases reported in the appendix, at the end of this lecture, is that of a female.—Ed.]

Dr. Duchenne and I know a gentleman who has been ataxic for more than twenty years. He has never manifested any intellectual disorder himself. But his father committed suicide, and his two sons have laboured under the most peculiar nervous affections. One of them, although of perfectly sound mind, is irresistibly impelled to shriek in a most extraordinary manner nearly all day; the other has had, and still has, singular muscular spasms. These are examples again of the transformation of nervous affections through hereditary influences.

I now pass on to the study of locomotor ataxy, when the disease is fully developed.

When children walk along a narrow plank or the edge of a boat, you must have noticed the peculiarity of their gait. In order to maintain their equilibrium, they take one step forward, stop, sometimes go backwards again, and incline their body to one side or the other, instinctively putting their arms out like a sort of balancing pole. In fact, their movements resemble those of an unskilled rope-dancer.

The gait of an ataxic patient is something like this. At the outset of the complaint, he staggers a little, especially as he gets up after having sat down for a long time. He rests on a stick or on the chair which he has just left, and he starts. As he takes the first step, the arm which does not rest on the stick leaves his side and oscillates like that of a rope-dancer, and his body inclines a little forwards. His walk is at first slow and uncertain, but becomes involuntarily hurried. Whereas in true paralysis, the leg is slowly lifted off the ground and is dragged along; in ataxy, the foot is thrust forward in variable directions, and comes down suddenly. Instead of the measured flexion of the knee-joint, which obtains normally, the flexion is sudden and followed by forcible extension.

When the disease is in a more advanced stage, if the patient does not rest on a stick, he throws his legs about with still greater disorder, and the inequality of his steps renders the loss of equilibrium still more imminent. Both his arms are then moved about like those of a rope-dancer, and his trunk itself is inclined or straightened according to the displacement of his centre of gravity.

This uncertainty and difficulty of progression do not prevent the patient from walking several miles on even ground, and he will often tire out persons free from any nervous affection. We had an instance of this in the case of a stonemason, who was admitted under me, September 18th, 1861. He had great difficulty in walking a few paces over the waxed floor of the ward, and yet on the previous day he had walked (almost without fatigue) from one end of Paris to the other.

When the disease, however, has made pretty considerable pro-

gress, the violence and irregularity of his movements soon exhaust the patient's strength, and he can scarcely walk a hundred paces before he gets out of breath, and is thrown into profuse perspiration.

There even comes a time when, although he still possesses muscular power, he cannot move a single step without falling down. If he be then supported by two persons under the arms, whilst he tries to walk, his legs move like those of a puppet, and are thrust to the right and to the left, forwards and backwards, with inconceivable disorder. From this time forwards he is obliged to keep in bed. The muscles of his trunk become affected also, and he can no longer sit up in a chair, unless he holds on to it with his hands, when his arms are not themselves implicated.

You can easily understand, gentlemen, how grave the prognosis must be in such cases. Death inevitably supervenes, and all the more quickly that sloughs form on the nates and about the trochanters, and that the suppuration to which they give rise rapidly exhausts the patient.

Instances, however, occur of patients who even at this advanced stage of the disease regain, sooner or later, some degree of motor power, and you had occasion to see this in the case of a man at No. 11 in St. Agnes ward. After having been for a long time compelled to keep to his bed, he improved so much that he was first able to get down his bed by himself, next to walk a few steps, resting on a companion's arm or taking hold of a chair or going from bed to bed, and later he could come up or go down stairs. This amelioration lasted several months, and I was indulging the hope that he would get well, when he was seized with homoptysis accompanied by all the signs of phthisis, which ultimately carried him off.

In the same ward, you can at present see another patient afflicted with locomotor ataxy and amaurosis, who after having been on several occasions compelled to keep perfectly quiet, can now walk by resting on a chair, and guiding himself with a cane.

When the disease has reached one of the stages which I have just described, the diagnosis is in general easy, even if the affection has been studied in books only. At the outset, however, great care is required, and few physicians, unless familiar with the neurosis, are able to recognize it.

In the early part of August, 1861, I was consulted by a chemist residing in a western province, who complained of some weakness of the lower extremities and the bladder. The lightning-like pains of which he also complained, and the deafness of one ear which I detected (in his case replacing diplopia or amblyopia) led me at once to suspect locomotor ataxy, and a more careful

investigation only confirmed my suspicions. For this I used a test which is of the highest importance, and to which I am desirous of calling your attention most particularly.

You have noticed already that, at an advanced stage of the disease, when the patient is in the dark, or when he voluntarily shuts his eyes, the uncertainty of his gait increases so much that he is absolutely incapable of moving a single step without falling down. This phenomenon, which is a symptom of very great value, manifests itself, although in a less marked degree, yet strikingly enough, from the very outset of the complaint.

The last patient, whose case I was relating, although complaining of weakness in the legs, which weakness did not really exist, yet walked without tripping, and maintained his equilibrium perfectly. As soon as he closed his eyes, however, he immediately staggered like a drunken individual, and would have dropped down if I had prolonged the experiment for some time.

The irregularity of the patient's walk, when his eyes are closed, is of later occurrence, and therefore of less diagnostic value than the next one which I am now going to mention.

If you ask an ataxic individual to stand up, and keep his feet closely applied together along their inner edges, he manages to do it with some difficulty when his eyes are open, even at an early stage of the disease. But when he shuts his eyes, he immediately oscillates and falls down, unless he be supported, or unless he opens his eyes and takes hold of a point of support, or, again, unless he makes considerable exertions to recover his equilibrium.

Thus, the walk of the patient whom I first mentioned presented little uncertainty only; but when his feet were closely approximated, he found it perfectly impossible to maintain his equilibrium on shutting his eyes. This sign, then, is of great value; and all the more so, that in paralysis nothing of the kind is observed. I have often had in my wards patients afflicted with hemiplegia, sequential to cerebral hæmorrhage, and sometimes also individuals attacked with general paralysis. I have made them walk and stand in your presence, with their eyes alternately closed and open, and you have been able to satisfy yourselves that they did not lose their equilibrium when their eyes were shut.

Every patient, however, who suffers from locomotor ataxy does not walk in the manner which I have described above. Thus, the patient at No. 23, in St. Agnes ward, who has double amaurosis as well, walks very much like a blind man. He carries a stick in his left hand, and in his right a small cane, with which he guides himself, whilst he walks *in a hurried manner*. A blind man walks, in general, in slow and measured steps, regularly balancing himself from right to left, but this patient constantly hurries forwards, and trots more than he walks, with a jerked step, oscillating when he stops. A blind man can remain per-

fectly motionless when he stands; an ataxic patient, on the contrary, loses his equilibrium, because his muscles are always in a state of exaggerated spasmodic contraction. In some exceptional cases, the patient's limbs are stiff when he walks, and his body moves all of a piece, as it were.

I was lately consulted by a patient whose intelligence was perfect, and who had mydriasis, but no strabismus. He had, besides, paralysis of the sexual organs, dating one month back, cutaneous anæsthesia, and what he termed paralysis of the lower extremities. He was constantly tripping, and dared not go out alone; when he walked he was obliged to take short steps only, otherwise his movements became disordered. He was not really paralysed, because his muscular power was still considerable, and he was only in the first stage of the disease.

At an advanced period of locomotor ataxy, spasmodic contractions are frequently observed, not only when the patient wills a regular movement, but even in the state of rest. In the latter case they consist in very powerful jerks of the limbs, and are an important symptom of this singular neurosis.

Patients then state that whilst they are walking, or even whilst they are merely standing, they feel as if the ground suddenly gave way beneath their feet. The cause of this is that the flexors of the limbs have been suddenly seized with spasm, and, overcoming the resistance of the extensors, have produced the sudden flexion of one of the lower limbs.

You may remember a woman lying at No. 23, in St. Bernard ward. When her legs were exposed whilst she was lying down, we could often see them shake and quiver with extraordinary violence. If, with both my hands, I encircled her thigh, I could feel the quivering of her muscles, whilst her foot moved with extraordinary violence and rapidity, without her knowledge and against her will.

Dr. Duchenne and I saw a patient at Montmartre suffering from well-marked locomotor ataxy, and who presented equally violent spasmodic movements.

In July, 1861, an old patient of mine, who had been suffering from this disease for more than twenty years, fractured both bones of one of his legs. In spite of the apparatus applied, the injured limb was constantly shaken convulsively, and the treatment considerably interfered with.

The pains, which I described in the first period of the disease, are usually, but not always, more intense when the disease is fully developed. They torture the patient, and extend to the trunk and upper limbs. Bodily fatigue, and the least moral emotions suffice to bring them on again.

They most frequently recur in paroxysms, that is to say, they show themselves for a few hours or a few days, every week or

every month, and then disappear. In other cases they are continuous, recurring from ten to thirty times in an hour, and deprive the patient of sleep for months and even years. We had an instance of this in the case of a picture-dealer who was in St. Agnes ward, and who was subject to such intense and frequently recurring pains, that his face always wore an expression of suffering. Belladonna and opium only gave him very transient relief.

Whether frequent or rare, these pains usually set in and go off suddenly. Sometimes, however, the patient is warned of their coming by some morbid sensation in the stomach or the genital organs. Thus a lady, who often consults me, is seized at intervals of two or three months with shooting pain in the lower limbs or the walls of the chest, sometimes preceded by epigastric *malaise*, sometimes by a dragging sensation about the region of the womb. It is a sort of *aura* which starts from those regions, and which ascends and descends to the spots which are suddenly seized with acute and transient pain. At other times, however, these pains set in of a sudden, unpreceded by any sensation, so that this form of *aura* may be after all due to a special susceptibility of the stomach and womb; and I am the more inclined to think so because the lady is liable to frequent attacks of gastralgia, and has suffered, for several years, from dropsy of the left ovary.

The premonitory anæsthesia becomes general after a time. The patient feels the ground imperfectly, and when to cutaneous anæsthesia is superadded the loss of muscular and articular sensibility, the patient can no longer feel the resistance of the ground, and if he shuts his eyes, he may, as Dr. Duchenne and I found out last year, have the sensation of being suspended in the air. Sometimes the patient fancies the ground is elastic, and that he is walking on India-rubber or on compressible balls, and this strange sensation persists even when sight can help him to correct his mistake.

Mucous membranes may also become anæsthetic. The patient at No. 2, in St. Agnes ward, suffers from anæsthesia of the mucous membrane of the mouth. He does not feel bodies placed in contact with his lips, sometimes drops the food which is between them, and cannot distinguish the temperature of what he eats or drinks. His teeth have lost their special sensibility, and cannot distinguish substances which are easily broken down from those that are not. The mucous membrane of his tongue obscurely perceives sapid substances, especially on the left side. The upper extremities are likewise affected sometimes, and the patient loses his sense of touch, occasionally also all muscular, osseous, and articular sensibility; but retains normally the sensibility to differences of temperature.

In some cases, and this is an important fact to remember, locomotor ataxy may be unattended with impairment of sensibility. I have myself seen a few cases of this kind, and several have been recorded by other physicians; as the one published by Dr. Oulmont, and the case you may have seen at the Lariboisière Hospital, in Dr. Hérard's ward. Dr. Lecoq has also published two analogous cases, in the *Archives Générales de Médecine*, 1861, in which no special pains had ever been felt, and sensibility had remained normal.

However exceptional these cases may be, I admit they are still highly important, as proving categorically that cutaneous and muscular anæsthesia are only secondary phenomena of the disease.

When the disease is fully developed, the various affections of the eye, which I mentioned as occurring at the outset, show themselves again, and may remain persistently. Thus diplopia, amblyopia, and amaurosis, as well as paralysis of the third or sixth nerve, may again be noted. Both eyes may be affected, although this rarely happens; whilst in other cases, as I have noticed several times, these various affections may be totally absent.

I have also met with paralysis of the fifth nerve, as shown by anæsthesia of the mucous membranes of the eye, nose, and mouth, and the skin of the face.

Anaphrodisia is often present, together with paralysis of the sphincters of the rectum and bladder. Yet, in three cases which I saw with Dr. Duchenne, the sexual power was unimpaired, although the bladder and rectum were seriously affected. In some cases the fundus of the bladder is paralyzed but not the sphincter, producing retention of urine; or the rectum is paralyzed whilst the sphincters of the anus act normally, so that obstinate constipation is the result. The retention of urine may be followed by grave consequences, as in cases of paraplegia; cystitis, for instance, may set in, and if the inflammation spreads upwards to the kidneys, death may result, preceded by symptoms of urinæmia or of pyæmia.

Locomotor ataxy, like many other diseases, is pretty frequently incompletely developed. Thus, at the outset, and sometimes for a period of several years, it reveals its existence by a few symptoms only, the significance of which may escape the observer. In one case, for instance, there will be merely paralysis of the muscles supplied by the third and sixth cranial nerves; in another, there will be more or less complete amaurosis, which, after having resisted every treatment, gets well spontaneously; or again, acute pains in the lower extremities will alone be complained of, which the patient compares to electric shocks, and which are so characteristic that, whenever they are mentioned to

me, I immediately suspect incipient locomotor ataxy. Indeed, since my attention has been drawn to this point, I have seen so many patients whom I at one time regarded as suffering from vague neuralgic pains, or from muscular rheumatism, present, from a few months to two or three years afterwards, the most characteristic symptoms of locomotor ataxy, that I now keep on my guard. I have often been able to find out by closely questioning patients affected with such shooting pains, that they also presented some of the premonitory symptoms of ataxy; such for example, as impotence and spermatorrhœa.

The *progress* of this disease is usually slow, and it may extend over a period of ten and even twenty years. A friend of mine has been ataxic for twenty years; and I am at present attending a Polish officer who has been ataxic since 1846, and who yet took a very active part in the Hungarian war of 1848. He can now ride his horse every day; and although he does not feel his stirrups (so great is the insensibility of his feet), he yet manages to sit his horse well through the great strength of the adductors of his thighs, and on one occasion, as I was testing his strength, he gave me very great pain by squeezing my hand between his knees. In some cases, however, the disease may run a rapid course, as in a patient from Saulieu, whom I had under my care, and in whom the disease became generalized in the space of six months. You yourselves saw an instance of this in the case of the stonemason who was in St. Agnes ward.

The *prognosis* in this disease is relatively of extreme gravity, for if in some cases it may remain stationary for a long time, it does not however get well. When speaking of the treatment, I will discuss the question whether it can be arrested.

I shall now endeavour to analyze the principal phenomena which characterize the disease, and attempt to give an idea of the defect of co-ordination of movement which constitutes the most striking symptom of confirmed ataxy.

In the act of skating, there is required a very remarkable co-ordination of all the movements of the foot, leg, and trunk; for as the impulse forward is given, the skater must keep in equilibrium on a single skate, that is to say, on a very thin blade of iron, placed vertically. He must put one foot down on the ice at the very instant that he lifts the other up. And when both his feet are together, learned combinations of muscular actions are necessary to enable the skates to avoid or get over an obstacle; he must often bend his body forwards, backwards, or sideways, and he must be careful to use his arms as balancing poles. Now the necessity for muscular co-ordination, which is strikingly marked in the act of skating, exists for all the movements of the body as well. Those which look the simplest require a precision, the difficulty of which we forget, owing to the habit we have

formed of executing them automatically. Indeed there is, in reality, no simple movement: when we flex our fingers, for instance, the flexors are not the only muscles which are called into play; but the extensors, which antagonize them, must also contract. For the performance of every movement there is required a common action of several muscles tending to the same end, and this common action, or muscular synergia, as it is termed, produces the harmony of movements. When this is at fault, defect of co-ordination results, and this constitutes one of the chief characteristics of locomotor ataxy, and of St. Vitus's dance.

In the majority of patients who are afflicted with ataxy, tactile sensibility diminishes, and is even abolished, especially in the sole of the foot and the skin of the leg. This anæsthesia extends sometimes to the trunk, although it gradually diminishes from below upwards. One kind of sensibility persists to the last, namely, that which takes cognizance of differences of temperature.

The anæsthesia may extend deeper than the skin, and affect the muscles and articular surfaces. The irregularity of the movements may be considerable, even when there is no loss of sensibility. At the end of August, 1861, I was consulted by an eminent barrister from Dublin, who had formerly been a patient of the illustrious Graves, and had lately been under the care of Drs. Corrigan and Carmichael. In his case sensibility was perfect, and yet the defect of co-ordination was so great that he was not able to walk unless supported by the arm of a companion. When both the cutaneous and deep sensibility, however, is lost, the inco-ordination of movements reaches its maximum.

I now proceed to discuss a very important physiological point, to which several eminent physicians have ascribed the principal share in the production of locomotor ataxy.

Sir Charles Bell observed the following case:—

"A mother, while nursing her infant, was seized with a paralysis, attended with the loss of muscular power on one side of her body, and the loss of sensibility of the other. The surprising, and indeed the alarming, circumstance here was, that she could hold her child to her bosom with the arm which retained muscular power, only so long as she looked to the infant. If surrounding objects withdrew her attention from the state of her arm, the flexor muscles gradually relaxed, and the child was in danger of falling."

Sir Charles, therefore, believed that muscles are supplied by nerves endowed with two distinct properties—the one giving muscular power, and the other muscular sensibility; and according to him, "muscular power is insufficient for the exercise of the limbs, without a sensibility to accompany and direct it."

Sight, however, can supply the absence of this sensibility, as shown in the above case. To the consciousness of exertion, Sir Charles Bell gave the name of *muscular sense;* but Gerdy, who perhaps was not aware of Bell's essay on the subject, suggested for the same faculty the name of *sense of muscular activity;* and in 1855 Dr. O'Landry published a memoir on "*The Paralysis of the Sense of Muscular Activity.*" For my part, I confess that I am by no means convinced of the existence of this sense, and I do not see that it is proved by the case recorded by the illustrious English physiologist.

An important distinction must be drawn between the consciousness of a movement which has been executed and the consciousness of the muscular contraction which performs the movement. When, after shutting our eyes, we execute, without effort, a pretty extensive movement, we are unable, even on paying the strictest attention, to feel the contraction of our muscles, although we may feel the movement communicated to the levers by the contracted muscles. This fact is so true, that when we ask an intelligent person, who knows nothing of anatomy and physiology, which is the seat of the movements through which the fingers are flexed or extended? he immediately points to the hand, and never to the fore-arm. It is only when the muscular effort is considerable, or kept up for a long time, that it is perceived where the contraction really occurs. Normally, then, we have no consciousness of muscular activity, but merely the consciousness of the movement itself, which is a perfectly different thing.

Another proof of this is the following:—If we make the hand, the fingers, or limbs of a healthy individual go through a series of passive movements, the extent and variety of these movements are perfectly appreciated by the person. But although his muscles are completely inactive, he is not conscious of this, but feels the movement which is performed, although he does not know by what means it is executed.

Every one may repeat these experiments, and will be then convinced that this so-called muscular sense of Bell, or sense of muscular activity of Gerdy, has no real existence in ordinary and normal contraction. Rest your elbow on a table, for instance, and flex or extend passively one of your arms, or the fingers of one hand. You will be perfectly conscious of these movements, even with your eyes closed; but the sensation which makes you affirm their existence, is partly psychical and partly dependent on a local impression.

Allow me, gentlemen, to explain my meaning. When I will a movement, I am conscious of its being performed, first, because everyday experience has taught me that our limbs invariably obey our will; and this is the psychical act which I mentioned

just now. But when I carefully analyze the impressions which are excited during these various movements, I perceive a very evident sensation, which is not seated, however, in the muscles of the arm. When my elbow is placed on a table, as in the above experiment, there is a sensation of pressure on the olecranon, which sensation is only felt in the skin. As the fore-arm is extended, a portion of the skin which covers its posterior and ulnar aspect will be in contact with the table; but when it is flexed, this latter contact ceases, while other points of the lower and posterior portion of the arm come in contact with the table, so that this double impression, entirely confined to the skin, tells me that I have completed a movement of flexion or extension.

The same thing happens when the hand is moved. If we shut our eyes whilst we move one hand, for instance, we feel in the palm, and in the palmar surface of the fingers, a sensation of dragging when the hand is opened wide, and a sensation of relaxation when the hand is closed, in addition to a special sensation in the joints themselves, which latter is always striking, and even painful, when we wake up from sleep. As to muscular sensations, they only exist when the contraction is extreme, or when the muscle is in a painful condition, as after a contusion or great fatigue, for example.

Understand me well, gentlemen, I do not deny muscular sensibility, as I have been said to do, but what is a very different thing—I deny the existence of a sense of muscular activity. Muscles are, indeed, endowed with an obtuse sensibility, as surgeons in their operations have found out thousands of times. This sensibility, which scarcely feels the incisions of a sharp knife, is very acute in cramp, in what is called muscular rheumatism, in inflammation of the muscular tissue, and after extreme fatigue; but from an abnormal state of sensibility we must not infer the existence of a physiological sensibility. Ligaments and articular surfaces become very painful in cases of arthritis or of sprains. The neck of the womb, in metritis, is sometimes exquisitely sensitive, and yet you are aware of the slight degree of sensibility to pain in a condition of health.

A healthy muscle, when galvanized, feels pain. If the biceps be pinched sharply, pain is felt, which, although not very acute, is perfectly distinct from the pain in the skin, and which must, therefore, be seated in the muscle. Muscles, then, are endowed with sensibility, but, I repeat, this sensibility is totally different from the sense of muscular activity, the existence of which I deny, as a non-psychical phenomenon. If, in the last experiment which I mentioned, the movements of the hand and arm be passive, instead of being spontaneous and active, the person experimented on will know perfectly, even with his eyes shut, that the movements have been executed. But he will be informed of this, not

by his sense of muscular activity, for his muscles shall have been inactive, but by the nature of the pressure made by another person's hand on his own, and by the sensations which he will feel in his skin and the vicinity of his joints. Cutaneous and deep sensibility, therefore, plays in this case a very important part, and this it is which regulates the movement.

It is this sensibility, that is to say, the impression made first on the skin, then on the deeper structures, and next on the articular surfaces, which enables us to appreciate the form, weight, and resistance of objects. If you lay the back of your hand flat on a table, and then place an object in your palm, you will at once appreciate, without making any muscular effort, part of its shape and weight; and this notion will at once tell you how much muscular power you shall need to take hold of and displace the object. The impression received will be responded to by your will, and your muscles commanded to act in proportion. But suppose, now, that the superficial and deep structures of your hand have become insensible. If you then successively place in your hand two objects of the same shape and colour, but of different weight and consistency, on trying to raise them in turn, you will either go beyond or stop short of the mark, and your movements will be marked by morbid irregularity. Yet your muscles will have retained their power, and what Bell, Gerdy, and Landry call muscular sense, or sense of muscular activity, will not be in the least impaired. Cutaneous and deep sensibility alone is affected, which is the monitor of the mind, and consequently of the will.

When a person, whose cutaneous and deep sensibility is lost, attempts to execute movements, he is like a deaf individual who tries to speak. The pitch of a man's voice is raised proportionately to the distance at which he wishes to be heard, but persons who are deaf, having no means of judging of the pitch of their voice, either raise it most inopportunely, or lower it so as to be inaudible. If I do not admit, then, the existence of this so-called muscular sense of Bell, or sense of muscular activity of Gerdy, I need scarcely add that I cannot admit Dr. Landry's theory, that locomotor ataxy consists in the loss of this sense of muscular activity. If you study this neurosis in its gravest form, and in its most advanced stage, I confess that you will find muscular sensibility and the consciousness of resistance and pressure gone. But in no instance is muscular sensibility deeply impaired, without the sensibility of the skin and articular surfaces being equally so; and I do not see why we should resort to a function and to properties, the existence of which is by no means proved, when we can interpret the facts in the simple manner which I explained to you just now.

But remember, gentlemen, that you may, on the one hand,

meet with patients suffering from ataxy, in a very advanced stage even, who have still retained their muscular sensibility, as in the case of the barrister from Dublin whom I mentioned to you, of three of my patients in St. Agnes ward, and of another patient who was under the care of my regretted colleague, Dr. Legroux. On the other hand, there may be muscular anæsthesia, and yet no locomotor ataxy, as in the case of a house-painter who was admitted into the St. Agnes ward in May, 1861. There was complete anæsthesia of the skin over his whole body; he did not feel pricking or pinching, but could distinguish differences of temperature, and when a vase filled with cold water was placed on his thigh, he complained of very disagreeable sensations. He did not feel violent pressure of his muscles; and when he contracted his muscles powerfully, he only knew of their acting because he had willed it, but he had no feeling of their doing so. The sensibility of his hands and feet was nearly perfect. Yet, in spite of this complete muscular insensibility, he walked naturally, even when his eyes were shut.

This case, which I studied with the greatest care, proves, therefore, that muscular insensibility, which necessarily implies the loss of the sense of muscular activity, does not suffice for producing locomotor ataxy, and there must, in my opinion, be superadded another element, to which I shall revert by-and-by; namely, *spasm*.

I may add, gentlemen, that in some cases, rather uncommon, it is true, even at a pretty advanced period of the disease, the patient complains of an extraordinary degree of cutaneous and muscular hyperæsthesia. In July, 1861, a practitioner of Rouen sent me a patient suffering from locomotor ataxy, in a very advanced stage. Both the patient and his medical adviser had been particularly struck with the exaggerated sensibility of the skin and the deeper structures of the limbs, so heightened, indeed, that the least contact or pressure gave unbearable pain. At the time when I examined the patient, these curious phenomena had passed away, but they had lasted several months, during which the irregularity of the patient's movements, and the difficulty he had in maintaining his equilibrium, were already considerable.

The following case, which I studied carefully, would seem, at first sight, to favour Dr. Landry's theory; but a more exact analysis of the case admits of its being interpreted in a manner contrary to those views.

On January the 26th, 1863, I was asked by Dr. Collongues to see, with him, a lady about 40 years old, who had been seized with left hemiplegia, about the middle of the year 1862. There had been at first complete loss of sensation and power of movement, without notable impairment of the intellect. By degrees the power of moving returned, and when I saw the lady she was

sewing with her right hand, and held her work in her left hand, the one which had been previously palsied. Sensibility was *completely abolished* in the arm and hand and the whole lower limb, except in the sole of the foot, which still retained a certain amount of *very obtuse* sensibility. Hard pinching of the skin, violent compression of the muscular masses, gave absolutely no sensation at all, nor did pressure of the articular surfaces against one another give rise to any.

Her hand could be opened out or closed, her forearm bent or extended, without her knowledge. Differences of temperature she perceived, however, perfectly. When I asked her to open out her closed hand, which I held in mine, she performed the movement; but although she *knew* that she performed it, she yet *did not feel it*. Even when she shut her eyes, she could do as directed. If, when her eyes were closed, I opened out her hand, and then asked her to extend her fingers, she moved them as if trying to do as she was bid, and if I told her that her hand was closed, although it was wide open at the time, she exerted herself to stretch out her fingers in an exaggerated and disorderly manner.

Without looking at them, she could bend each of her fingers in turn, into her palm, although her movements in so doing lacked precision.

In order not to drop any object which she might be holding, she was obliged to keep her eyes on her hand, and she squeezed the object with unnecessary force. If she closed her hand forcibly, when empty, she did it with such violence that her nails wounded her palm.

When she was engaged in conversation, and her attention could not thus be kept fixed on any object she might be holding, she had recourse to a peculiar stratagem, in order not to drop it. She pressed it against her chest, which had still retained its sensibility, and she could thus rectify, and in some degree measure, the contractions of the muscles of the forearm which moved the hand.

By a *constant exertion* of her will, she could supply the absence of sensibility of the skin, muscles, and articular surfaces. Normally, however, no *manifest* exertion of the will is needed to enable a person to hold an object in his hands, even when his attention is diverted from it, as shown by the familiar circumstance of a man carrying a stick or an umbrella without his attention being always directed to it.

The muscular power of the patient was *nearly normal*. The left forearm, when bent on the arm, resisted extension nearly as much as the right; and the patient opposed as great a resistance also to the forcible abduction of her left arm from the trunk as when the same experiment was tried on the right side.

The movements, by which she alternately closed or opened her hand, were marked by the same irregularity as in the most confirmed cases of ataxy. Thus, instead of closing her hand by at once bending her fingers and thumb, she bent each of them in turn, in a strange and disorderly manner. This became considerably more marked when her eyes were shut, and was less so when she kept looking at her hands, whilst moving them. In the alternate movements of flexion and extension of the hand, it was easy to perceive that the antagonistic muscles no longer regulated those movements which were therefore more extensive than they should have been.

At night, the patient did not know where her left arm was, unless it was in contact with some sensitive portion of her body; it might hang out of bed without her being aware of it.

When she walked, she threw her left leg and foot forward exactly as an ataxic individual. She was obliged to keep her eyes on that leg, or else she would have fallen down. She knew, however, that her left foot rested on the ground, from a sensation of resistance in the hip-joint, and a very faint sensation in the sole of that foot. But this two-fold impression was not sufficiently distinct to allow her to walk in the dark. At night she did not know the position of her left leg unless it was in contact with the right.

This case is interesting in many regards. First of all, the age of the patient and her previous history exclude all possibility of an hysterical affection, and therefore all idea of deceit, which nervous women unfortunately practise in too many cases. Secondly, it is very evident that the case never had the aspect of locomotor ataxy. The disease had set in with sudden hemiplegia, probably the result of an extravasation of blood into the brain. There had never been the characteristic pains of ataxy, and no impairment of the sight. The anæsthesia of the skin, the muscles, and joints, had occurred suddenly; and this fact excluded all idea of ataxy. Now the defect of co-ordinating power was as great as it could possibly be, and no one would have hesitated to call the patient ataxic, had she presented the symptoms which usually precede and accompany locomotor ataxy. Cases such as this, if they were frequent, would favour Dr. Landry's theory; but as I have already said there are a good many instances of locomotor ataxy, and in which cutaneous, muscular, and articular sensibility is thoroughly preserved, and I have drawn your attention to some of them in my own wards.

On the other hand, recall to mind the case of that young man, suffering from diphtheritic paralysis, who was in the St. Agnes ward, in January, 1863, and that of a young woman, similarly afflicted, who was in the St. Bernard ward, in the beginning of the year 1862. In both those patients the sensibility of the feet

and skin of the legs was singularly diminished, in fact, nearly abolished. Pressure of the muscles was not perceived, movements of the knee and ankle were not felt, and yet the gait of those patients was not in the least like that of ataxic individuals, but merely of persons struck with paralysis. They dragged their feet with difficulty along the floor, and the uncertainty of their walk was no greater than that which results from ordinary muscular paralysis. When they were asked to shut their eyes, they could still walk, although with increased difficulty.

You see then, gentlemen, that if one case seems to tell in favour of Dr. Landry's theory, other, and more numerous cases, decide against it. In the case of the lady mentioned above, hæmorrhage into the cerebellum, or softening of that organ, might be suspected from the suddenness of the seizure, and the form assumed by the first symptoms which manifested themselves. If so, the case would come under the category of *cerebellar ataxy*, to which attention has been drawn by Drs. Bouillaud, Hillairet, and Duchenne.

The circumstance that passive movements of his limbs are not perceived by the patient, has by some been regarded as a characteristic symptom of locomotor ataxy; but this is a grave error, which is disproved by clinical observation. I admit that in hemiplegia due to cerebral hæmorrhage or softening, passive movements of the palsied limbs are perfectly perceived, and that the perception is often attended with pain, provided, however, that the patient be not comatose. I will add, that compression of the muscles, in such cases, often gives rise to pain and to a sensation of cramps on the paralyzed, and not on the healthy side. But this does not happen in most cases of paraplegia (and it is especially with paraplegia that locomotor ataxy may be confounded). In July, 1861, I saw a young lady from Bernay, who had complete paralysis of the lower limbs. Pressure and pinching of the skin were feebly felt. When her eyes were shut, she felt imperfectly if her leg was touched, but she was not aware of my pressing the muscles of her calf, although they were then thrown into violent and convulsive contractions. If, after extending her leg, I held her foot in my hand, and then forcibly adducted, abducted, flexed, or extended the limb, or pressed strongly the articular surfaces against one another, she felt no sensation whatever. At night she did not know the position of her limbs. These were really the symptoms ascribed by Dr. Landry to loss of the sense of muscular activity, and yet the case was one of paralysis, and not of ataxy. Dr. Landry has been led into error by the fact, that in the last stage of locomotor ataxy, anæsthesia supervenes; but it is a symptom which belongs to most cases of paraplegia.

In locomotor ataxy which has not yet reached its final stage,

even when the irregularity of the movements clearly points to the disease, muscular and articular sensibility may be preserved, as in the cases of the superior officer and the Dublin barrister, which I have already mentioned. The uncertainty of the gait may be considerable, especially when the patient shuts his eyes, although he may feel the ground perfectly, and although sensibility may not seem in the least impaired.

The fact that the patient's gait becomes much more uncertain when he shuts his eyes, is not a symptom either of the loss of the sense of muscular activity. The same thing occurs in perfect health, and happens as much, after all, in a clear case of paraplegia as in one of locomotor ataxy. Any one of us here will walk badly on shutting his eyes; and even when sure of meeting with no obstacle, he will walk in a peculiarly hesitating manner. The well-known bet of the Versailles lawn has clearly proved that no one can walk, with a handkerchief before his eyes, from one end of the lawn to the other, without getting into the gravel-walks. This is a proof that walking must of necessity be guided by sight, and that what has been called the sense of muscular activity is not sufficient for this purpose.

A blind man who walks on a pavement, however much he may be used to do without his sight, is yet obliged, in order not to deviate from his course, to use a stick, which brings him back into the normal direction which he is constantly losing.

Now, in order to explain the simultaneous existence in ataxic patients of extreme incoordination of movements and very slight diminution, or even perfect preservation, of cutaneous and muscular sensibility, the existence of a *deep* or *common sensibility* (*cœnesthesia*) has been assumed, the impairment of which is said to account for the phenomena. The author[1] of a recent critical review on the subject defines this new kind of sensibility thus:—"It is the very distinct perception (although almost unnoticed from its being so continuous) which we all have of the presence of our organs, their volume, weight, shape, situation, and relations. From all the points of the organism there constantly ascend to the nervous centres an infinity of sensitive currents which, luckily for us, are not noticed by the encephalon, but the *interruptions* or *irregularities* of which strike us forcibly as soon as they occur."

I confess, gentlemen, that I cannot conceive a common, unnoticed sensibility, which reveals itself to us only when it is *interrupted*, in other words, when it ceases to exist. The very men who admit its existence, confess that physiology cannot teach us what it is, and that pathology alone can do so. Yet the

[1] Dr. Axenfeld: "Des Lésions atrophiques de la moelle épinière." (*Archives énérales de Médecine*, août et octobre 1863, p. 486.)

few examples which have been given in illustration have not convinced me. They all consisted of cases of individuals "who could not tell positively the position of their limbs, unless they saw them, and who suffered merely from a very slight impairment of tactile sense, limited to a circumscribed region of the trunk." The same author cites the cases of two women, observed by himself, one of whom "was obliged to note the place where she lay her hands before going off to sleep, in order to be sure of finding them in the dark." This case would appear of little value to me, even if the observer had noted the condition of the *articular sensibility*, which he forgot to take into account.

Remember, however, that this so-called *deep* sensibility should not be confounded with the *organic* sensibility which nobody denies. This latter manifests itself, only when it is exalted in pathological conditions. The stomach, for instance, which in health performs its functions silently, makes its presence felt in bad digestion; and other organs, which are normally insensible, acquire, under the influence of what is called inflammation, an exaggerated sensibility, and become the seat of the most acute pain.

I now pass on to the *differential diagnosis* of locomotor ataxy. It most frequently attacks the lower limbs first: the patient believes that he is seized with paralysis, and his mistake is shared by some medical men, very excusably indeed when there is real paralysis of the bladder and rectum at the same time, with a sense of circular abdominal constriction, tingling of the lower extremities, &c. In order to avoid this mistake, however, the patient's muscular power need only be tested, and the absence of real paralysis will thus become evident.

Tumours of the cerebellum give rise to a form of ataxy. Dr. Hérard has lately published, in the *Union Médicale* (t. iii. 1860, p. 230), a very remarkable case of the kind. The patient had lost the power of associating, combining, and co-ordinating the movements which make up the complex act of locomotion, standing, &c. There was no paralysis of sensation or motion, general or partial, direct or crossed. So far, the symptoms of the case resembled those of locomotor ataxy, since the latter disease need not be accompanied by paralysis. A distinction, however, was afforded by the extreme frequency of vomiting, in Dr. Hérard's case, which symptom, according to the interesting researches of Dr. Hillairet, accompanies cerebellar diseases. Moreover, the patient had never presented the premonitory symptoms of genuine locomotor ataxy, namely, the characteristic pains in the limbs and the trunk, the disturbances of vision, and the various forms of local paralysis, &c.

I need not go into the differential diagnosis of ataxy and the general paralysis of the insane and chorea: these two last

diseases are attended with such characteristic symptoms that a mistake seems to me impossible.

When speaking just now of cerebellar ataxy, I should have mentioned Professor Bouillaud as having been the first who experimentally and clinically studied the effects of injuries to the cerebellum. As far back as 1828, and later in 1847, in his "Nosographie Médicale," my learned colleague described *the various co-ordinated movements* which injuries of the cerebellum impaired; namely, walking, standing, and the maintenance of equilibrium. For a detailed account of his researches, I must refer you to some lectures delivered by Professor Bouillaud, and which have been recently published by Dr. Auguste Voisin.[1]

§ 2.—Pathological Anatomy of Progressive Locomotor Ataxy.—Relation of the Lesions to the Symptoms.—Nature of the Disease.—Treatment.

In his treatise on "L'Electrisation localisée," in which he was the first to give the clearest and most complete description of the symptoms of progressive locomotor ataxy, Dr. Duchenne (de Boulogne) says not a word of its *pathological anatomy*. At least, he gives one case only in which he had occasion to seek, after death, for the alterations which might characterize the disease; and in that case, the subject of which was an individual who, in 1858, died in one of Dr. Nonat's wards at the Charité Hospital, "*the encephalon and spinal cord, on the most careful examination, presented no anatomical lesion appreciable to the naked eye.*[2] These negative results did not support the theory which Dr. Duchenne had, *à priori*, broached of the nature of the disease. Indeed, reasoning from the fact that, since the beautiful researches of Flourens and Bouillaud, the cerebellum was looked upon as the seat of the faculty of co-ordinating movements, Dr. Duchenne suggested that, in locomotor ataxy, the defect of motor co-ordination, which constitutes its primary phenomenon, must be "*necessarily dependent on some structural or functional lesion of the cerebellum.*" Secondly, when he took into account the order of sequence, and the progress of the symptoms, he was led to believe that the central morbid process which gave rise to the symptoms, generally began in the motor nerves of the eye and the corpora quadrigemina, from which they extended to the superior peduncles of the cerebellum, and lastly to that organ itself.

Now that a pretty good number of cases have been recorded, showing that in progressive locomotor ataxy it is the spinal cord which is diseased, and nearly always a limited portion of the

[1] *Union Médicale*, 18, 25 et 28 juin 1859.
[2] "Traité de l'Électrisation localisée," p. 608.

cord, the dorso-lumbar especially, and very rarely the cervical, whilst the cerebellum is not notably affected, Dr. Duchenne has given up his first theory. The anatomical appearances found in ataxy are confined, as a rule, to the posterior columns of the cord and the roots which issue from them; it is only in exceptional cases that the antero-lateral columns are implicated as well. These appearances consist sometimes in a kind of grey degeneration, and sometimes in a gelatiniform and translucent condition, in a diminution of consistency, or in a state of induration, called *sclerosis*. In the greater number of cases the posterior columns are sensibly diminished in size, but in some very rare instances the volume is increased. The alterations of the posterior roots are proportionate to those of the cord, that is to say, they are most marked in the roots which are connected with the most diseased portions of the cord.

With regard to the microscopical appearances, allow me to quote the following extract from a recent memoir of Dr. Axenfeld,[1] in which are summed up the observations made by a good many authors :—

"In the *white matter* of the posterior columns, which has now become yellowish or grey, are seen scattered nerve-tubes, pale, shrunken, or varicose, sometimes reduced to their neurilemma only or filled with granular contents, a few still retaining their *cylinder axis*. On the other hand, the connective transparent substance (*neuroglia* of Virchow), the blastema in which these tubes are imbedded, has become fibrillated, and presents amidst a large quantity of amorphous granules, a smaller quantity of elongated nuclei, and a smaller one still of cells (perhaps the nuclei, or at least most of them, belong to the nerve-sheaths). Corpora amylacea, also, are met with in variable quantity, distinguishable by their usual reaction with tincture of iodine. Lastly, the blood-vessels are considerably developed, and their thickened walls, composed of several layers, are incrusted with a deposit of fatty granules.

"In the posterior cornua of the *grey matter* the same alterations are found, but less markedly. The reddish tint of this part is due to the injection of its capillary network, and occasionally its tint is darker, blackish, owing to the presence of numerous granules of pigment. The nerve-tubes in these cornua are sometimes destroyed, and the nerve-cells altered in shape, although in general both the tubes and cells are normal.

"The changes noticed in the posterior roots are the same as those of the corresponding columns; and they are the same again in the diseased portions of the bulb, pons, optic nerves, &c.

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[1] Dr. Axenfeld : "Des Lésions atrophiques de la moelle épinière." (*Archives générales de Médecine*, août 1863, p. 224.)

"On the whole, all these alterations clearly point to *atrophy* of the nervous tissue."

The first two cases in which I had occasion to note these pathological appearances are the following. The first one was published by Dr. Bourdon, under whose care the patient was at the time of his death.[1]

Mr. W., a man of letters, aged 38, was admitted March 22, 1861, under Dr. Bourdon's care, into the Maison Municipale de Santé. He has formerly led a rather dissipated life, and has often had painful emotions and known real grief. When about 25 years old, he had well-marked epileptic fits, probably due to his excessive use of absinth, for the fits disappeared after two years, and on his giving up drinking absinth.

The symptoms of his present complaint first showed themselves about six years ago. At first slow and uncertain in its course, the disease afterwards kept on the increase, especially for the last six months, during which time he has had a good deal of trouble and undergone great fatigue. The first symptoms were uncertainty in the movements of the limbs, and mere stiffness when walking, followed by some difficulty in going up and particularly down a staircase, in spite of the most powerful exertion of the will.

Eighteen months ago, weakness of sight supervened, with occasional diplopia. For the last six months he has had slight incontinence of urine with marked diminution of sexual power. Since this last date, he has for the first time been seized with sub-occipital pain extending to the nucha and shoulders, becoming less violent, though not entirely removed, when he was lying down, but intensified by the standing or sitting posture, so much so as to be unbearable, and to compel him to lie down.

For some time now, he has been free from this pain. The left upper eyelid drops slightly; there is external strabismus of the left eye, the pupil of which is markedly more dilated than that of the right eye. In addition to the diplopia, which depends on the paralysis of the third nerve, there is a diminution in the range of distinct vision. Thus, although he can see perfectly a person when very close to him, he cannot distinguish his features when standing at a very short distance. The upper limbs and trunk are not affected, either as regards sensation or motility; but the lower extremities are strangely diseased. The act of walking is extremely painful and laborious, from the limbs moving with considerable stiffness and irregularity. The patient cannot control his movements, and is unable to come down a staircase

[1] See "Études cliniques et histologiques sur l'Ataxie locomotrice progressive." (*Archives générales de Médecine*, novembre 1861.)

without falling. He keeps looking at his feet when he walks, as if sight were needed for guiding them; and, indeed, when he shuts his eyes he cannot move a single step, unless he can rest on, or guide himself by, a piece of furniture. At night he has occasionally been obliged to go on all fours when he wanted to move a short distance from his bed. Yet, it is easily ascertained that his muscles have preserved their contractile power, for if an attempt be made to flex his legs on to his thighs against his will, he resists it with great vigour. When he has placed himself in equilibrium, he can stand very well, and can even bear a heavy weight on his shoulders. He is conscious of the contractions of his muscles, and knows when they are compressed. Even when his eyes are shut, he performs any movements which he may be asked. Tactile sensibility and sensibility to pain are perfectly normal in the lower limbs, even in the soles of both feet.

He has never had any boring pains, like those described by Dr. Duchenne (de Boulogne). For the last month he has been completely impotent; he has never had spermatorrhœa, or the rapid emission during coitus to which Dr. Trousseau has drawn attention. Professor Trousseau himself noted all the above symptoms, when he saw the patient in consultation.

The gastro-intestinal affection, which set in ten days after his admission, resisted all treatment. Intense and incoercible diarrhœa came on; the motions were passed involuntarily, vomiting and hiccup next set in, and the patient died of exhaustion, his intellect being unaffected up to the last. The *post-mortem* examination was made thirty-four hours after death. The weather was rather cold, and there were no signs of putrefaction. The cerebrum, cerebellum, and pons varolii were injected in certain spots only, and looked healthy. The spinal cord, on the contrary, was deeply diseased. The examination was carried on by a distinguished micrographer, Dr. Luys, and the results are therefore highly important.

1. The *dura mater* (of the spinal cord) was very vascular in its whole length, and of a dark-red hue. It was also very markedly thickened in its upper portions, and somewhat œdematous. There was no trace of old exudations.

2. The spinal *pia mater* was likewise abnormally injected, more markedly so over the lower third of the cord and along its posterior columns. In those points, in fact, it strongly adhered to the posterior columns, and, like them, had a yellow tint. It could not be separated from the cord without tearing away some of the nerve-substance.

3. The *posterior columns* were the parts most peculiarly diseased. They looked like two transparent vitreous bundles, in some parts of an amber-yellow colour, and in others of a reddish-yellow tint, according to the greater or less degree of vascular

injection. Their consistency was diminished, but they were not diffluent, and, moreover, there was no solution of their continuity, By spreading out their fasciculi with a needle, they could be easily traced for some distance.

This *degeneration* of the posterior columns was most marked in the lumbar region, but it extended to the dorsal also, being exactly limited to the space comprised between the posterior cornua. It gradually diminished, and finally disappeared in the brachial region, although, even on a level with the upper portion of the bulb, the white matter near the grey commissure could be seen to present appreciable traces of a similar degeneration.

This special discoloration of the posterior columns was owing to the transformation of their *nerve-tubes*. Most of these, indeed, had been destroyed; and there remained of them nothing but their empty sheaths, the walls of which were applied against one another. The nerve-tubes which had not wasted away completely, still retained their cylinders, with this difference, however, that these cylinders, instead of having smooth edges and a ribbon-like aspect, and of being of the faintest pale-yellow colour, were now rough, jagged, and of a yellow tint like that of amber. Amidst the nervous elements, were scattered capillary vessels in large numbers.

4. The *lateral columns*, with the exception of a very slight and superficial yellowish discoloration in their lowest portions, were perfectly healthy in all their extent, from the lowest to the highest limits of the cord.

5. The *anterior columns*, in the lumbar region, were less thick, and less firm to the touch than usual; their colour was normal, and differed totally from that of the posterior columns.

6. *Grey matter.* In the lumbar region, and for the lower fourth of the cord, it had lost its consistency, especially in its central portion. Its fibres were all more or less ruptured in places, although a few could still be traced; and in those points the shape of the anterior and posterior cornua was still perfectly distinguishable. Thus, on examining transverse sections of the cord, networks of cells extending from the posterior to the anterior cornua could be seen in some sections, whilst in others made one millimetre higher up or lower down, only ruptured fibres could be found, accumulation of fatty granules, and a shapeless detritus. Even in those degenerated portions, however, all the *nerve-cells* had not totally disappeared; a certain number of them could still be seen, with their prolongations, but most of them, either those of the anterior or of the posterior cornua, of the grey substance of Rolando, or of the intermediate regions, were shrunken, with jagged edges, and covered with a larger quantity than normal of granular pigment; in a word, they were undergoing a process of *involution*. The capillaries of the grey matter

were considerably enlarged. The capillary plexus had evidently been the seat of partial and transient congestions, for in those parts where the grey substance was less consistent than usual, there were found amorphous and diffused deposits of hæmatin, pointing to antecedent congestive processes.

7. *Posterior roots.* The nerve-fibres were unfortunately examined only in that part of their course from the posterior columns to the ganglions, not beyond the latter. (A) First, as regards the condition of *the ganglions.* All those of the lumbar region were of larger size than normal, and unusually red and vascular. Their consistency was not diminished, and their enveloping membrane was notably thickened. On section, in addition to enormously dilated capillaries, there were found evident traces of old congestions, with diffusion of hæmatin. Moreover, the *ganglionic cells,* instead of being only partially covered by a few granules of brownish pigment, as they normally are, were sprinkled over with reddish-yellow granulations. Some of the cells were shrunken, with lacerated edges; others, on the contrary, were voluminous, pale, discoloured, almost spherical, and looking very much like fat-cells, with which they might have been confounded, were it not for remaining traces of the old nuclei, and the vestiges of their nerve-tubes still adhering to their walls. A few of these ganglionic cells still retained their normal relations to the nerve-tubes which surrounded them.

The ganglions of the lumbar roots were the only ones which presented these lesions, limited to portions of ganglions.

(B) *Roots.* The nerves of the cauda equina presented very characteristic appearances. Instead of their usual cylindrical form, firm consistency, and whitish colour, they were flattened out, ribbon-like, and looked like strips of parchment which have long been macerated in water. Those which came from the anterior columns were transparent and of a greyish colour, whilst those which were connected with the posterior columns had a vitreous aspect, and were of a uniform yellowish tint. Large vessels, more numerous than in health, accompanied the nervous fasciculi which were connected with the posterior columns.

All the nerve-fibres which connected the ganglions with the posterior columns were of the same yellowish colour. The nature of the degeneration was the same, and produced the same collapse of the walls, and the same amber-yellow aspect of the cylinders, when these were still present.

The posterior roots were diseased in the lumbar region only; in the dorsal region, they by imperceptible degrees resumed their normal aspect, and in the upper regions of the cord they were not in the least modified. Thus the roots of the *glosso-pharyngeal, vagus, auditory,* and *trigeminal* nerve, were not appreciably affected.

8. *Anterior roots.* Generally speaking, the anterior roots were infinitely less affected than the posterior. In the lumbar region these roots were less firm than usual, greyish, and transparent. The nerve-tubes were not very sensibly altered, and in most of them continuous cylinders could be seen, unbroken and contained in a normal sheath. The intervening nerve substance was very markedly diminished, so that these nerves, which are usually of large size and of a white colour, looked, in consequence of this atrophy, like the nerves found in the grey portions of nervous centres, and which are without white substance.

In the dorsal region, the anterior roots looked normal again, as well as higher up in the cord. The roots of the *spinal accessory*, and of the *facial*, looked healthy on both sides, as well as the roots of both *hypoglossal* nerves.

The *abducens oculi*, however, and the *motor oculi*, on both sides, looked remarkably altered. The latter had the aspect of greyish cords somewhat œdematous, and shrunk to nearly half their usual size. They readily gave way when, by gentle pulling, an attempt was made to remove the brain from the cranial cavity. The *abducens oculi* was similarly affected on both sides, although to a less extent. They were both of diminished volume and consistency, and of a greyish colour. The walls of their nerve-fibres were collapsed, and in some their contents (white substance and cylinder axis) had been completely reabsorbed. Numerous capillaries were interlaced round the nervous fasciculi.

On following up, in the grey matter of the fourth ventricle, the trunk of the abducens oculi as far as its real origin, large vascular trunks were found in a series along the course of the primary fibrillæ of this nerve, which they must probably have compressed considerably.

The roots of the *fourth nerve* had the same colour and consistency.

The next case which I am now going to bring under your notice is that of a patient who died in Dr. Vigla's ward, and I owe the following details to Mr. Dumontpallier, who assisted Mr. Sappey in making the post-mortem examination:—

Pothel, aged 55, formerly a messenger, has enjoyed good health previous to his present ailment. There has been no nervous complaint of any kind in his family. He has been a messenger since the age of 18, and has thus been exposed to changes of weather and to great fatigue, which did not, however, affect his general health. He has never had syphilis.

In 1849 he felt shooting pains always attacking the same parts of the trunk or lower limbs, and recurring in paroxysms about every fifteen days for half an hour. At first he paid little attention to them, on account of their short duration and the distant intervals between the paroxysms, and he went on with his usual

occupation. These pains, however, by degrees returned at shorter intervals.

Two years afterwards, in 1851, he became impotent; in 1852 his gait became a little uncertain, and by the end of the year he could neither walk nor even stand, and he had to be carried. He noticed nothing peculiar about his eyes or bladder.

In 1861 he was admitted, under Dr. Vigla's care, whilst suffering from bronchitis, and the presence of pulmonary tubercles was diagnosed.

Dr. Duchenne (de Boulogne) having been requested by Dr. Vigla to examine the patient, ascertained from him the above details. He found, besides, that cutaneous and muscular sensibility was perfect everywhere, that partial movements were performed with normal power when tested whilst the patient was in a *sitting* or *lying* posture, and that, nevertheless, the patient could not maintain his equilibrium when standing, nor *take a single step unless supported by two attendants.* The want of harmony in the movements was such that the patient could not walk with the slightest regularity. He left the hospital after a two months' stay.

In April, 1862, he was again re-admitted with symptoms of galloping phthisis. With regard to his locomotor ataxy, the following notes were taken:—His *muscles* are very much wasted, but not more in one place than in another. Cutaneous and muscular sensibility is considerably diminished in the right leg. When he is pinched, the painful sensation is only perceived three seconds afterwards. In the left leg cutaneous and muscular sensibility is more acute than in the right, but is still abnormally diminished except on the posterior aspect of the limb. Pinching is faintly perceived.

In both thighs the various kinds of sensibility are normal. In the soles of the feet the sensibility to tickling and pressure is considerably diminished, especially on the right side. Sensibility to pinching has almost entirely disappeared.

The sensibility of the upper limbs and trunk is everywhere normal. Partial movements are still executed, although very feebly in consequence of the wasting of the muscles. The patient is incapable of standing or even of sitting. There is no affection of the bladder or of the eyes.

Six days after the above notes were taken, he died of his pulmonary disease.

The *post-mortem* examination was made twenty-four hours after death, and Mr. Sappey examined the spinal cord under the microscope. The brain was well formed, of normal consistency, and without a trace of injection. Successive sections of the organ showed it to be healthy throughout.

The cerebellum, pons varolii, and medulla oblongata were also healthy.

The cervical and dorsal portions of the spinal cord were of normal size, colour, and consistency; the lumbar portion alone was slightly diminished in size.

On dividing the latter transversely at its upper limit, the surface of section of the posterior columns was seen to be of a greyish hue, evidently pointing to an alteration of these columns, which were still however of normal consistency.

The anterior roots of the lumbar portion of the cord were normal; the posterior, on the contrary, were very considerably atrophied, this atrophy being peculiarly striking when a healthy cord was placed by the side. The roots were thus shown to have lost about two-thirds or three-fourths of their normal size. Their aspect was also considerably modified; they were not white, but of a reddish-grey colour, looking pretty much like bundles of capillary blood-vessels. Besides, they did not project sufficiently from the surface of the cord, as they issued from it, but spread out like delicate ribbons of scarcely any thickness.

Under the microscope, the nerve-tubes of the posterior roots were seen to have lost a considerable portion of their medullary substance, although some of them looked still normally full, and contained a cylinder axis. Those that were reduced in size were contracted in one point, swollen out at another; in a word, they were very irregular. In a great many, the medullary substance had completely disappeared, so that they looked constricted here and there. In some, again, vestiges of the medullary substance reappeared at long intervals. Where it was completely absent, the tubes, when examined with a power of 400 diameters, had a filiform appearance, without, however, presenting a perfectly regular contour.

As the nerve-tubes were unequally altered, the progressive series of their degeneration could be traced out. Those that were normally filled with medullary substance, accounted for the retention of sensibility in several portions of the integuments; whilst the empty or nearly empty tubes explained the impairment of sensibility in the lower limbs.

The rapid *post-mortem* alteration of the cord unfortunately prevented an examination of the posterior columns.

Now, gentlemen, do you not find singular contradictions between the symptoms of progressive locomotor ataxy and the pathological appearances found in the cord?

In a disease essentially characterized by disorders of motility, and in which the loss of sensibility is relatively of secondary importance, since this property may be more or less unimpaired, one might have expected to find the *anterior* not the *posterior* columns diseased, according to the physiological doctrines professed by Mr. Longet. Yet the absence of disease in the anterior columns accounts for the absence of real paralysis, and the defect

of co-ordinating power may be physiologically explained by disease of the posterior columns. This want of co-ordination, as I have several times told you before, is not due to anæsthesia, for this may be transitory only, or very slight in degree, or even be completely absent.

As to the persistence of sensibility, notwithstanding the grave lesions of the posterior columns and their corresponding roots, microscopical examination has proved that there still remained in the degenerated nerve-tissue a variable number of healthy nerve-tubes, from which it has been concluded that these healthy tubes extend their sphere of action beyond their own area, and thus supply the place of those which have disappeared.

The anatomical fact itself cannot be disputed, but the inference from it is very questionable, and as Dr. Axenfeld has judiciously observed: "The very small number and the diminished size of the fibres which have escaped disorganization, their absence even in some cases, do not admit of such an interpretation. And even had slight anæsthesia existed at one time, and been unnoticed by the patient, there would always be a very singular and unforeseen disproportion between the imperceptible disturbance of the function and the profound alteration of the organ which is supposed to discharge that function." [1]

The above conclusion is still more disproved by the results of experiments performed by Brown-Séquard, Türck, Philippeaux, and Vulpian, showing that sensibility has remained normal after the complete destruction of a portion of the posterior columns included between two transverse sections. It is true that similar experiments, repeated by Leyder and Rosenthal, gave contradictory results. But these contradictory statements of savants of acknowledged merit are only an additional proof that as regards the functions of the spinal cord and the nervous system in general physiology has not said its last word. At all events pathological observation seems to favour the opinion advocated by Brown-Séquard, Türck, Philippeaux, and Vulpian, that the posterior columns of the cord directly and immediately influence the co-ordination of movements. Besides, Dr. W. Gull has drawn attention to the fact that Todd regarded the posterior columns as the centre of the faculty for co-ordinating voluntary movements.[2]

I will not dilate further on this still obscure question of pathological physiology, but will at once tell you what my idea is of the *nature of this disease*, and what place I am inclined to give it in nosology.

When first I spoke to you of *progressive locomotor ataxy*, I

[1] Eléments de Pathologie Médicale de Requin, art. Ataxie Musculaire, Paris, 1863. p. 683.

[2] Guy's Hospital Reports, t. 4, p. 169, 3rd series, 1858.

looked upon it as belonging to the great class of *neuroses*, and one of the writers who continued Requin's work, Dr. Axenfeld, regarded it in the same light. At that time the pathological anatomy of the disease was very little known, but I still maintain my opinion, although numerous *post-mortem* examinations have now revealed the existence of more or less grave organic lesions of the cord. I base my opinion on clinical observation, on the nature of the symptoms which point to disturbances essentially due to an affection of the nervous system, on the absence of fever, on the evolution of the symptoms, their variety, and the mobility of some of them. As to the material lesions, the existence of which seems to be incompatible with the idea of neuroses, those lesions, if Todd's theory be accepted, confirmed as it is by the experiments of Brown-Séquard, Philippeaux, and Vulpian, account in some measure for one of the phenomena of progressive locomotor ataxy, and the most prominent I admit, namely, the defect of co-ordination of the movements. On the one hand, however, these lesions by no means account for all the symptoms; and, on the other, it would be wrong to regard the disease as dependent on them, since they are only a consequence and an effect, as I shall explain presently.

First,—understand me well on this point. When I say that the disease is not dependent on the presence of the material lesions found, I mean only those which can be detected by our present means of investigation. For, as I have stated to you on numerous occasions, I cannot conceive a functional disturbance without a special corresponding modification of the organ which discharges that function. This may be more or less transitory, and it frequently does not alter the structure of the organ any more than an overcharge of electricity alters the structure of the glass or the metal of a Leyden jar, and it therefore remains perfectly unknown to us. Now, as regards progressive locomotor ataxy, the fact that the lesions on which it is said to depend are not so constant as it has been positively affirmed, is an argument in favour of my opinion. In some cases, although ataxy had been present for several years, and been attended with the most distinct and characteristic symptoms, skilful anatomists have not been able, either with the naked eye or with the aid of the microscope, to make out the slightest alteration of the posterior columns and roots. These cases are rare, and very exceptional, I admit, but a single case is sufficient to strengthen the opinion which I maintain, and no one can reject the following, which was observed by Dr. Gubler whose competence in such matters is well known.

A man about 44 or 45 years old, had, twelve years previously, suffered from the characteristic pains of locomotor ataxy. After lasting three years, these pains left him, and were followed by paralysis of the third nerve on the left side, which

persisted until death. Amblyopia next set in, at first affecting the left eye only, but after a time implicating the right also, and ending in double amaurosis, with atrophy of the optic disc. About five or six months previous to his admission into the hospital, he complained of a sense of weakness in the limbs, and sometimes of difficulty in maintaining his equilibrium. Later, he noticed inco-ordination of the movements of his lower limbs, which rapidly increased, and extended to the upper limbs; and, lastly, of loss of sexual power.

He died on October the 16th, 1863, of an attack of small-pox, in the course of which he was seized with general paralysis, belonging to that class of paralysis which occurs in acute diseases, and which has been so well described by Dr. Gubler.

A fortnight before his death, the patient was examined by Dr. Duchenne, who pronounced the case a typical one of locomotor ataxy. Partial movements were performed by the patient, with considerable power still, and yet the inco-ordination of his combined movements was such that he could neither walk nor stand, and he could not use his upper limbs except with difficulty. Although his muscles were not very developed, there were yet no local depressions on his limbs which pointed to the existence of progressive muscular atrophy. Besides he presented no symptoms of that affection.

In this case, gelatiniform degeneration of the posterior columns of the cord and atrophy of the posterior roots were fully expected; but to the great surprise of all, these lesions were not found after death. The *spinal cord* was, it is true, *generally injected;* but this, at the most, only accounts for the general paralysis which set in at the last. The *optic nerves* had a gelatinous aspect, and under the microscope their tubes were seen to be atrophied; the motor oculi on the left side was slightly atrophied. These were the only anatomical lesions found, lesions which also occur pretty frequently in locomotor ataxy. Microscopical examination of the cord and its roots, made by Drs. Gubler, Luys, and Duchenne, detected no alteration in the posterior columns and roots. Dr. Duchenne found on the contrary, on examining transverse sections of the anterior lumbar and cervical roots, that about one-third of their tubes had disappeared.

Just now, gentlemen, I told you that the *post-mortem* appearances found in progressive locomotor ataxy were not the cause, but the effect, the product of the disease.

When we examine the lesions found, we are struck with three facts:—First, the atrophy of the nerve-tissue of the posterior columns and the corresponding roots; secondly, the development of cellular tissue, or if you prefer the term now generally adopted, the hypertrophy of the *neuroglia;* Thirdly, the vascularity of the diseased tissues.

The atrophy of the nerve-substance is the most striking phenomenon. It evidently does not belong to the category of those simple forms of atrophy which takes place in organs condemned to prolonged physiological inactivity, and which is the counterpart of hypertrophy due to an excess of functional activity. But although it is the most striking phenomenon, this atrophy is only a consequence of the pathological *evolution* of the cellular element, which, by developing itself, has crushed the nerve elements contained within its areolæ; and this abnormal development of the cellular tissue is itself dependent on the increased vascularity of the tissues.

Now is this increased vascularity sufficient to characterize inflammation, and are we from its presence to conclude that progressive locomotor ataxy is only *a variety of chronic myelitis?* If so, how are we to explain why this myelitis is always so exactly limited to the posterior columns of the cord and to the roots issuing from them; and particularly why, during life, it is attended with symptoms differing so much in their form, course, and changeability, from those common to all varieties of myelitis.

This abnormal vascularity of the posterior columns of the cord, which is again observed in the motor oculi and optic nerves and the tubercula quadrigemina, seems to me a consequence of frequently repeated congestions, analogous to those which we see during the patient's life affecting the conjunctiva. This membrane, as I mentioned to you already, gets injected in the interval between the paroxysms of pain, simultaneously with the occurrence of contraction of the pupil which is sometimes carried to an extreme degree. Generally, however, this injection disappears on the supervention of pain, especially of pain in the head, whilst the pupil dilates more or less at the same time.

These congestive phenomena show themselves in other diseases acknowledged to belong to the class of neuroses, such as hysteria, asthma, and Graves's disease (exophthalmic goître); and they belong in my opinion to the same category as those which in his experiments Professor Cl. Bernard produces at will by dividing the sympathetic.[1] They point to some disturbance in the functions of that nerve, of which we neither know the nature nor the cause.

It now remains for me to speak of *the treatment of progressive locomotor ataxy*. I am unfortunately compelled to be brief on this point, for the great number of remedies which have been tried against this disease, is a proof of their inutility and of the impotence of medicine. If as yet, however, we do not possess any means for curing this affection, or even for arresting its progress,

[1] "Leçons sur la Physiologie et la Pathologie du Système nerveux." Paris, 1858, t. ii.

we can still, in certain cases, modify and moderate some of its symptoms, and thus procure some alleviation. Above all, we are to avoid remedies which have proved not only useless, but dangerous. Thus we should reject bloodletting absolutely, whether general or local; purgatives also, which, when often repeated, act in the same way; revulsives, cauteries, moxas, or setons, which, by causing irritation of the skin, may bring on the special pains of ataxy in the spots where they are applied.

Flagellation, however, used methodically and in moderation, in some of my cases, has diminished the pains.

Dr. Duchenne (de Boulogne) also states that he has often known *cutaneous faradization* diminish the cutaneous and muscular anæsthesia which, at an advanced period of the disease, aggravates the want of co-ordination of the movements; and the result, he adds, has been great improvement in the manner of walking.

In order to calm the pain which is sometimes very acute, I have recourse to *belladonna* and *spirits of turpentine,* which I prescribe alternately, for ten or fifteen days successively, in gradually increasing doses.

Lately, Professor Wunderlich has published several cases in which the progress of the disease has been apparently checked by the internal administration of *nitrate of silver.* Drs. Charcot and Vulpian, who tried this remedy in their turn, have reported favourably of it.[1] Since then, other cases have been published in favour of it; but, unfortunately, cases may be opposed to these, in which the nitrate of silver failed completely. I myself have very often prescribed it both in my private and hospital practice, and although I, *à priori,* expected some good from a remedy which I had found useful in a good many neuroses, I must confess that in locomotor ataxy it has not fulfilled my expectations.

Hydropathy and *sulphur baths* are indicated in this disease as general modifiers; and above all, the patient's strength should be supported by all the means in the physician's power.

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[1] Charcot et Vulpian: "De l'Emploi du Nitrate d'Argent dans le traitement de l'Ataxie progressive." (*Bulletin général de thérapeutique,* 1862.)

APPENDIX BY THE EDITOR.

The following well-marked cases of progressive locomotor ataxy, which have come under my observation at the National Hospital for the Paralysed and Epileptic, will be found to illustrate most of the points discussed in the above Lecture :—

Case 1.—W. R——, aged thirty-five, a bricklayer, married, and the father of five children, two of whom are living, the youngest being two years and a half old. He has never had gout, rheumatism, or syphilis ; in fact, he has enjoyed very good health all his life, and his present illness is the only one he remembers of a serious nature. He has always been abstemious, and has lived well, but has had to do very hard work, to walk very long distances, and be frequently exposed to wet.

In 1847 he was run over by a cart, the wheels of the cart, which was not loaded at the time, going across his loins, bruising them severely. He was carried to an hospital, for he could not walk, and there he remained for ten days as an in-patient, but was discharged at the end of that time perfectly well.

In March, 1857, whilst daily engaged in some very hard work, in a damp place, and after having got repeatedly wet, he was seized with pain of a rheumatic character in both his knees, the right knee being considerably worse than the left. He noticed also that his toes were numb, those of the right foot more so than those of the left. About the same time, or very shortly after, there was numbness of the tips of the fingers of both hands, more marked on the left side. He could not grasp objects so easily, and could not undo a button which he did not see. He had no pain down the spine or in the head. He next complained of fatigue after short walks, and of a peculiar jerking, spasmodic movement of the right leg when walking—a sudden *catching up*, as he terms it, perfectly involuntary, and as if he were walking on springs. In spite of these sensations, he went on with his usual occupation as a bricklayer, and in June, 1857 (three months after the last date), he began to see double ; his friends also told him that he squinted—he thinks towards his nose. From the commencement of his illness he had more frequent calls to pass his urine than when in his usual state of health ; and when he was fatigued by any exertion, or attempted to lift anything heavy, his urine dribbled from him involuntarily. The bowels were regular.

The pain in his knees went on increasing gradually, and was always worse in the evening after a day's work. He felt growing weaker every day, and he became troubled with unpleasant *nervousness*, as he terms it ; for whilst he could easily walk up planks placed obliquely so as to form an inclined plane on the scaffoldings, he felt *too nervous* to walk down the same planks, and was obliged to slide down them.

In October, 1857, feeling much worse, he was compelled to give up work, and on applying at St. George's Hospital, was admitted an in-patient. Shortly before then, being late one morning, and hearing the clock strike, whilst on his way to his employer's place, he wished to run, but found he could not, and that he must walk slowly and cautiously. At the time of his admission he had a painful sensation as if the base of his chest were constricted by a broad bandage and his respiration impeded. During his stay in the hospital he also noticed that he was obliged to have recourse to sight for guiding his movements. When in bed, he could not tell the position of his limbs under the bed-clothes, unless he rubbed one knee against the other. Once being alone at dusk, in the water-

closet, he fell down on attempting to rise from the seat, as he was unable to see his legs and guide their movements. He was discharged in about six weeks (Nov., 1857), no better than he was before, and until Sept., 1861, he had no medical advice. He kept a small shop in which he moved with great difficulty; and on one occasion, being left alone in the dark, he could not strike a light owing to the unsteadiness of his hands, and he had so little feeling in them, that he squeezed the matches so hard as to break them. He remained in his chair for three hours, unable to rise or move in the dark, until a light was brought in accidentally.

Some time after he had left St. George's Hospital, but at what precise period he cannot remember, he became affected with occasional intermittent pains of a shooting character, which he himself compares to electric shocks. These pains came on in paroxysms, and attacked both legs, from the hip to the toes. The arms also were, after a time, liable to similar attacks. At first slight, they gradually increased in intensity, and were at their maximum in 1861.

He was carefully examined in February, 1864, and the following notes were taken of his case. He had been attending the Hospital for Paralysis and Epilepsy during the previous eighteen months as an out-patient, but was in February admitted an in-patient, under Dr. Ramskill's care.

He is a tall, spare man; complexion pale, with a sallow tinge; slaty discoloration of the edges of the gums in the upper and lower jaws (probably due to the nitrate of silver which he has been taking for months). Intellectual faculties unaffected. Articulation distinct; no stammering; no difficulty in pronouncing certain words or certain letters. The muscles of the face act perfectly. Hearing good. No ptosis; slight internal strabismus of left eye, and diplopia only when he looks over the left shoulder. Sight itself good; no amblyopia; both pupils contracted to the size of pins' heads; no injection of the sclerotic or conjunctiva. Respiration natural; when asked to breathe deeply and quickly, the respiratory movements still remain regular. Pulse very weak and compressible. Heart-sounds normal. Appetite very good; digestion easy. Bowels regular; no constipation. Urine healthy; no albumen; no sugar; no dribbling; no retention. No pain in the spine, not even on hard percussion; no sensation now of a circular band constricting the base of the chest.

On exposing the lower limbs of the patient, they look fairly nourished, and do not suggest the idea of paraplegia. The muscles of the calf are prominent, and exhibit no signs of wasting. The limbs do not feel colder than the rest of the body. *Sensibility:* (*a*) Tactile sensibility obtuse, for although he can tell the exact spot where he is touched, there still elapses some time before he perceives the contact; in other words, there is a retardation in the conduction of tactile impressions to the sensorium. (*b*) Differences of temperature are immediately perceived; (*c*) and painful sensations, such as pinching and pricking, are acutely and immediately felt. He, besides, complains of the same paroxysmal shooting pains down both legs, causing spasmodic jerking flexions of the knee-joints. (*d*) He feels, but not acutely, a powerful electro-magnetic current; and (*e*) whether his muscles contract under the influence of such a current, or in obedience to an effort of the will, he declares positively that he is conscious of muscular contraction taking place; hence there appears to be no loss of muscular sense. *Motility:* When lying down on his back he can move his legs perfectly, and when asked to extend them with all his force, they cannot be flexed by another person, thus showing that his extensor muscles have retained all their power. On the other hand, if he flexes his knee or foot, it requires considerable exertion on the part of another in order to extend the flexed limb forcibly, thus showing that the flexor muscles have retained all their strength. If now he be asked to rise, the efforts which he makes are peculiarly striking. He is obliged to take hold of some neighbouring object, and his legs seem to shake under him. He cannot remain long in the standing posture without resting on a stick. If he be made to close

his eyes he soon loses his balance, and is obliged to open his eyes to save himself from falling. His gait, when walking, is most peculiar, and differs from the ordinary shuffling of paraplegia. His feet are everted and apart from each other; he lifts his leg well off the ground in the first stage of progression, but in the second stage, before the limb has completed its semi-oscillation, he suddenly extends it in a quick, jerking manner, and lets it drop heavily on the ground, from the heel to the toes. He always puts the heel down first, and with great force. His eyes are kept fixed on his lower limbs, and there is evidently all the time an excessive and fatiguing exertion of his volitional power. He stops abruptly short before he can turn round. He prefers an uneven surface to a smooth one for walking upon, and he can go upstairs better than he can come down, taking care to place the whole length of the foot flat on the stairs. From the extreme attention he is obliged to pay to all the movements of his limbs, and the fatigue consequent thereon, he is averse to walking, and gets easily tired.

During the paroxysms of shooting pains there seem to occur reflex spasmodic movements of the lower limbs, but on tickling the sole of the foot at other times, no reflex contractions of the muscles of the legs can be produced. The patient is not, however, troubled with spasms or jerks of the legs when in bed at night.

In both his upper extremities there is the same sensation as in the legs, of numbness, with occasional flying, shooting pains. The left arm is more affected than the right, the reverse of what obtains in the lower extremities. The impairment of sensibility in the right hand is clearly shown by his being obliged to wear a handkerchief round the head of his walking-stick, so as to increase the area of contact, for without this precaution the stick is apt to roll out of his hand. The muscles of both arms are fairly nourished, and they contract with great force. When he stretches out his hand, however, to take hold of anything, there is a certain amount of unsteadiness, of hesitation, as it were, in the movement, showing a deficiency in the proper co-ordinating power of the arm. He has great difficulty in undoing the buttons of his shirt, but there is no loss in the hands of muscular sense, for he can grasp and hold in his hands any object, however small, even when his eyes are perfectly closed.

He left the hospital at the end of May, much better than when he came in; but the want of co-ordination of the movements of his lower limbs, although less, was still strikingly marked. Since then he has not presented himself at the hospital.

Case 2.—J. A——, a shipwright, aged forty, married, the father of six children, the youngest of whom is eleven months old. There is no history of paralysis in his family. He has himself never had gout or rheumatic fever. More than twenty-five years ago he had simple chancres, not followed by secondary symptoms. Fourteen years ago he had intermittent fever on the coast of Africa, and about the same time he became affected with slight shooting pains, which he believed to be of rheumatic character, and occasionally attacking the lower limbs. Six years ago he had in Bombay a sunstroke, followed by fever, which lasted two or three weeks. On his voyage home shortly after this, in 1858, he noticed a peculiar affection of his lower limbs, which although it could not be termed weakness, made him stagger and reel like a man under the influence of liquor. This peculiarity in his walk was very strikingly marked on his landing at St. Helena, and made him the butt of many jests from his friends. At that period his sight was not affected; he had no incontinence of urine even on exertion. The soles of his feet felt numb, however, and he had the sensation of a pad interposed between them and the ground. He suffered occasionally also from paroxysms of shooting pains in the lower limbs, but not very acute; scarcely so indeed as to attract his attention much. The unsteadiness of his legs during progression increased gradually, and became considerably marked between two and three years ago (1861, 1862). His bladder was affected for

the first time then, for he was obliged to pass his water immediately on feeling the want, or otherwise his urine dribbled away involuntarily. His arms were next attacked; the tips of his fingers felt numb, and he could not undo the button of his shirt-collar, because he did not see it, for he could undo the button of his shirt-front, which he did see; and he could not feel a sixpenny-piece in his pocket. When at work he had apparently suffered no loss of muscular strength in the arms, for he could deal as heavy blows with his hammer as in previous years, but there was some amount of unsteadiness in the movements which he performed with the arms. He thus volunteers the statement that he had some difficulty in hitting a nail right on the head, for his hammer often went by the side of it.

These symptoms went on increasing by degrees, and in the year 1863 he had nocturnal incontinence of urine; was not able to tell the position of his limbs when in bed, under the bedclothes, and discovered that he could not walk in the dark.

He became an in-patient of the Epileptic Hospital in 1864, under Dr. Ramskill's care, when I took the following notes of his case:—

He is a tall, thin, but healthy-looking man. Intellect unaffected; articulation normal; face symmetrical; no twitching of muscles of face or lips; hearing good; sight perfect; no strabismus; no ptosis; pupils normal, not contracted; respiration normal; deep breathing performed regularly. He has never had the sensation of a tight band constricting the base of his chest or his waist. Circulation feeble; pulse regular, but weak, 75; heart-sounds normal at base and apex; appetite fair; bowels regular, not costive; urine feebly acid, contains no albumen, no sugar. The same weakness of bladder exists as already mentioned. No spontaneous pain in any part of the spine, and none elicited by hard percussion; no deviation of the spine. The lower extremities look thin, but, according to the patient's statement, they are not more so than they have always been; in other words, they have not wasted. There are subjective sensations of cold in the legs, but to the touch they do not feel cold. There is numbness of the soles of the feet, but no tingling in the toes. Tactile sensibility is obtuse; the patient can tell the exact spot where he is touched, but there occurs an interval of two and three seconds, and sometimes more, between the moment at which he is touched and that when the impression is perceived. Differences of temperature and pricking he immediately perceives. He complains of sharp shooting pains in the limbs, passing through their whole length from the hips to the toes with considerable rapidity. These pains come on in paroxysms, at variable intervals, and are influenced by wet and damp weather which intensifies and aggravates them. He has no idea of the position of his limbs when he does not see them, but, strangely enough, he can execute with them any movements he is told. Those movements are more clumsily performed, however, than when he looks at the limbs, and he afterwards points very inaccurately to the direction in which the limbs are lying. When in the sitting posture he moves his legs with perfect freedom and ease; kicks with force; flexes, extends, and rotates the limb well, and successfully resists all attempts made by another person to flex or extend his leg against his will. With all this muscular power, his mode of progression is strikingly unsteady, and suggests the idea of paralysis. He has extreme difficulty in rising from his chair, and in starting on his walk. His gait is tottering; he lifts up his leg well from the ground, but then throws it about in a wild, disorderly way, incapable of controlling or regulating its movements. The whole limb is straightened out forcibly, and the heel comes down on the ground first and heavily. He cannot stand with his eyes closed, for he immediately loses his balance, oscillates, and falls down like a lump. When he walks, his eyes are kept fixed on his legs, but he cannot even then, with the help of his visual sense, restrain the wildness and disorder of their movements. He cannot walk without a stick, or without holding on to neighbouring objects, and from the immense exertion of his voli-

tional power he cannot walk any distance, gets easily tired, and is every few minutes obliged to stop and rest himself. There have never been reflex movements, jerks, or spasms of the legs at night in bed. None whatever can be excited on tickling the soles of his feet.

Both upper extremities look well nourished. The patient complains of numbness, chiefly in the tips of the fingers, but he can tell at once the exact spot where he is touched when his eyes are closed. He has occasional shooting pains along the ulnar side of both forearms, extending to the ring and little fingers. He complains of loss of power in the arms; but he can grasp with considerable force, and lift heavy weights. His movements are unsteady, and somewhat clumsy. When asked to hammer down a nail, his hammer more frequently goes by the side of it than hits it. When his eyes are closed, he cannot accurately tell the difference in the weight of various objects placed in his hands.

June 23rd.—The patient's condition is somewhat improved. He totters less when he walks, and he does not throw his legs about with the same degree of wildness and uncertainty; in fact, the inco-ordination of his movements is less. There is still great weakness of the sphincter vesicæ; and he often wets himself from the involuntary escape of urine. His hands do not feel so numb; and although he does it clumsily, and is a long time about it, he still contrives now to button or unbutton his shirt-collar without seeing it. The paroxysms of shooting pains recur at the same variable intervals in all the four limbs, but oftener affect the ulnar side of the forearms, extending to the ring and little fingers. The electro-muscular contractility of his lower limbs is perfect, but their sensibility to magneto-electric currents is considerably below par. His sexual power has not diminished; he is often troubled with erections.

Oct. 6th.—The patient is considerably better in some respects. His general health is excellent, and he has gained flesh lately; his lower limbs are not so thin as before; his calves feel cold to the touch, however, and he complains of a sense of great cold in them. He is subject to the same paroxysms of shooting pains, which have not increased in severity. The tactile sensibility of his thighs, legs, and feet is completely gone; he cannot feel a mere touch at all, not even a deep, prolonged pressure; pricking he feels acutely still. His manner of walking has improved nevertheless; he can walk across the whole length of the wards without a stick; with a stick he staggers less, and he can walk longer distances than before, always keeping his eyes on his legs. The same sensation of numbness exists in the upper limbs, not worse than before, and the movements of the hands are less clumsy.

Sept., 1865.—The patient left the hospital a few months after the above notes were taken, in the same improved condition as is there recorded; and although he was heard of from time to time, he was not seen till the present date. He has lost ground considerably in the interval, and is now very emaciated. He has lately been harassed by excruciating paroxysms of shooting, and occasionally boring pains, which, affecting him both by day and by night, have allowed him no rest. Within the last six months he has on three separate occasions been seized with epileptiform convulsions, attended with loss of consciousness. His sight has failed considerably; he can now read for a short time only, because his sight gets very misty, and he is constantly troubled with black spots before his eyes, as if twisted bits of thread were floating before them. He has no diplopia, and no strabismus; his pupils are smaller than they used to be, and when he faces an open window his right pupil contracts to a less degree and more slowly than the left. His lower extremities are almost completely anæsthetic, and the inco-ordination of his movements is much more marked than when he was an inmate of the Hospital. He has so much difficulty in restraining the wildness of his legs when he walks, that he dislikes the attempt, and never moves beyond his house.

Case 3.—C. Y——, aged forty-three, coach joiner, a widower, and the father

of eight children. No history of paralysis in his family. Previous health excellent; no diathesis of any kind; never exposed to wet; has always worked in dry places. In May, 1862, as far as he can recollect, he noticed that he was unable to wash his face unless he was supported by another person, or could lean against a wall; for as soon as he closed his eyes he lost his balance, staggered, and nearly fell. When walking, however, he could feel the ground perfectly, and there was not the slightest numbness of the soles of his feet. About three months afterwards (in August, 1862), without any assignable cause, he was suddenly seized with pain of a shooting, darting character in both lower extremities, passing through the limbs from the hips downwards with the rapidity of lightning. The first attack of pain lasted, with intermissions of a few minutes only, for about thirty hours. It then left him as suddenly as it had set in, but recurred two or three days afterwards for a few hours only. At that time he had no pain whatever down the spine. There was nothing abnormal with his sight: no squinting; no impairment of vision. From that period, however, he became subject to paroxysmal attacks of these peculiar shooting, lightning-like pains, at variable intervals of from a few days to two or three weeks. He had no boring, deep-seated pain: the pain he felt was superficial, exactly like the shock from a galvanic apparatus, and it was attended with jerks and startings of the affected limb. The lightest touch of the hand, mere contact of his clothes, rendered it more intense. It began in various parts of the limbs, sometimes in the knee, sometimes in the ankle, and sometimes in the toes; and from the first spot affected, it flew to other portions of the limb, occasionally, however, remaining persistently fixed for hours in the same spot. He had also the sensation of a tight band constricting the lower part of his abdomen. His bowels were not confined. The bladder was not affected at that time. Very shortly after the first attack of shooting pain he noticed some numbness of both his lower extremities, which became more and more marked with every recurring paroxysm of pain. About the same time, also, his walk became unsteady, his gait tottering, and he was obliged to look at his legs when walking. In the beginning of 1863, in addition to the above symptoms, weakness of the bladder supervened. He could not retain his urine as soon as he felt the desire to pass it, but was compelled to satisfy the want instantly, and even then he often wetted himself. He was not a widower at the time, and noticed a gradual diminution of sexual power also. Within the last six months all traces of virility have completely disappeared.

Present state, May 30th, 1864.—The patient is a thin, pale, ill-nourished, middle-sized man. Intellect unaffected. Articulation normal. Hearing good. Sight good, with the exception of occasional muscæ volitantes; both pupils are contracted to the size of pins' heads, but are easily dilated by atropine. There is no redness of the conjunctiva, no injection of the sclerotic vessels, no paralysis of any muscle of the eyeball, and no ptosis. Appetite not good. Bowels always regular. The urine contains an excess of phosphates, but no albumen or sugar. Micturition not more frequent than normal, and he very seldom wets himself now, although he is still obliged to satisfy the want as soon as it is felt. He has no pain whatever down the spine. The sensibility and motility of both upper limbs are perfect and normal. The lower extremities alone are affected. Their aspect, however, is that of well-nourished, healthy limbs; according to the patient's statement, they have not in the least diminished in size since the commencement of his illness. To the touch they are of the same temperature as the rest of the body, and there is no subjective sensation of cold in them. The patient can distinguish differences of temperature, and he distinctly feels pinching, pricking, &c. But his tactile sensibility is very obtuse: when his eyes are closed, he cannot spontaneously perceive mere contact, although, when his attention is directed to it, he becomes conscious of the sensation, but is even then unable to localise it accurately. He has a sensation of numbness in both legs, and is unable to tell their position if he does not see

them, when in the lying posture; yet, strangely enough, he can move them at will, although with much greater unsteadiness than when he can in some degree guide his movements by sight. The electro-muscular sensibility is considerably below par; strong currents, which make him wince and give him pain when passed through his upper extremities, are scarcely felt by him when his lower limbs are acted on. The electro-muscular contractility of the limbs, however, is not diminished, for their various muscles contract with normal force and energy. When lying down he can raise his limbs, flex, and extend them at will with force and rapidity, and he can successfully resist all attempts at bending his limbs made by another person when he keeps them fully extended; but as he gets up, and attempts to walk, his legs look strangely feeble. He cannot walk at all without the help of a stick, and although he keeps his eyes fixed on his lower limbs, watching their movements, his gait is peculiarly tottering and unsteady. It does not seem to him as if he were walking on springing boards, but he throws his legs about as if he could not measure the amount of strength necessary for executing the intended movement. He takes short steps only, and after wildly throwing his limb forwards, he suddenly extends the leg on the thigh, and brings the foot heavily down from the heel to the toes. He cannot turn round quickly when walking, but stops abruptly before he can do so. If he be asked, whilst standing, to close his eyes, he immediately loses his balance, oscillates from side to side, and would fall unless he opened his eyes, and laid hold of some neighbouring object. He is still liable to the shooting pains described in his previous history.

Sept. 7th.—He complains of numbness in the tips of his fingers, which he first noticed about two months ago. This numbness is more marked in the right hand. The paroxysms of shooting pains have lately recurred more frequently, although they have not increased in severity. They chiefly attack the left lower limb. He also complains of a sense of tightness in the abdominal muscles which to the touch feel tense and rigid. The sensibility of his lower limbs is in the same impaired condition as previously recorded. Their motility has, however, improved considerably; his movements are much less unsteady, he throws his legs much less wildly about, and is not obliged to be constantly keeping watch over them.

Case 4.—J. W——, optician, aged 41, married, the father of eight children, of whom five are living and three are dead. His father died at the age of 29, of consumption: his mother died when giving him birth. A first cousin of his has epileptic fits.

Previous health.—He has always lived well and occupied healthy rooms. More than 20 years ago, he had gonorrhœa and gonorrhœal ophthalmia, but never chancres. He has been subject for many years to rheumatic pains down his back and across his loins. In other respects, his general health has been excellent. His present ailment began about eight or nine years ago, whilst he was labouring under an attack of influenza, with shooting pains, *like toothache*, affecting both his lower limbs, generally along their posterior aspect, and very rarely their anterior. These pains came on in paroxysms almost every day, either in the daytime or during the night. They lasted a variable number of hours, and then disappeared spontaneously; they caused jerks and starts of the limbs, the muscles of the calf and ham contracting most energetically, as if a powerful galvanic current were being transmitted through them. He had, at the same time also, pain about the lumbar region of the spine—in the muscles of the loins. For about four or five years the peculiar paroxysmal pains just mentioned were the only symptom of disease that attracted his attention. He had no tingling in his toes; no sense of numbness, but his legs felt heavy, particularly when walking down hill. His walk, however, after this interval of time, became peculiar; he rolled about a great deal, and it seemed to him as if he were *walking on india-rubber*. If he walked by the side of a friend, and did not think of his legs, they

showed a disposition to go their own way, and kick his neighbour or tread on his toes. He is perfectly certain that, at that time, he could walk in the dark.

About five years ago, in 1859, on getting out of bed one morning, he found that his *right* upper eyelid had dropped, and that his *right* eye was turned *outwards:* he had also double vision. On the preceding day he had had a severe attack of shooting pains in his lower limbs. Within four months, and after a prolonged stay at Margate, his right eye got well, but ever since then his right pupil has been persistently contracted. He further volunteers the statement that when he has been comparatively well and perfectly free from pain, his pupil dilated a little. On his return from Margate he was considerably better. His pains were less severe, recurred less frequently, and, in fact, left him entirely for the space of three weeks. At the end of this time they returned, however, with renewed intensity on his suffering from an attack of diarrhœa, and, since then, he has been rarely free from them. Within the last 18 months, there has been diminution of the expulsive power of the bladder, and his water had to be drawn off on two or three occasions. A new feature of the disease also showed itself about that time; he found that he could not walk in the dark. He could feel the ground well enough, but his feet slipped from under him. During the day, he was not obliged, he says, to keep his eyes fixed on the ground or on his legs.

In December, 1863, he applied to the National Hospital for the Epileptic and Paralyzed. He had then slight strabismus divergens of the *right* eye; his right pupil was contracted, and for the preceding three weeks there had been loss of power of focal adjustment of the *left* eye. He also complained of shooting pains affecting both upper limbs, and chiefly felt along the ulnar side of both fore-arms, and extending to the tips of the little fingers.

He became an in-patient of the hospital, under Dr. Ramskill's care, in June, 1864, and shortly afterwards I took the following notes about him:—"He is a thin, sallow, middle-sized individual. His memory is very good, his intellect in all respects unimpaired. His emotional excitability is, however, abnormally heightened; his spirits are very low and depressed, and he has frequent inclination to shed tears. He has no pain in his head. His hearing is very good, although he has lately complained of tinnitus of the *left* ear. There is no strabismus of the *right* eye, no dropping of the *right* upper eyelid, such as have been recorded in his previous history; but within the last four days there has been complete ptosis of the *left* eyelid. The right pupil is contracted to the size of a pin's head; the left is about three times the size of the right. There is no injection of either eye. Vision with the right eye alone is good, but when he raises the left upper eyelid with his fingers, and looks at an object in front of them, he sees it double and one image is higher than the other. There is very slight but appreciable strabismus divergens of the left eye. During the paroxysms of pain, to be mentioned presently, the *left* side of his forehead is covered with perspiration, whilst the right side remains dry. There is some tremulousness of the muscles of the lower lip and chin, probably due to the emotion of the patient.

His appetite is fair: his bowels are costive. His circulation is very feeble: the heart sounds are normal. He has no pain down the spine, no sensation of a circular band constricting the base of his chest or his abdomen, but he complains of a sense of tightness limited to the left hypogastric region. There is weakness of the *bladder*. He is obliged to satisfy the desire of making water as soon as he feels it, and, even then, in spite of all possible haste, he wets himself frequently. His urine is feebly acid, somewhat cloudy, but clears up when heated. For the last eight months, he has entirely lost all power of erection, and before then he had for a long time noticed a gradual diminution of sexual power. He never, at any time, had spermatorrhœa.

Lower extremities.—The patient declares that his legs have got much thinner lately, but only as part of his general emaciation. There is slight adematous fulness of both ankles and dorsi of feet. He has no tingling, no pins and needles

in his toes, no formication, no sense of cold in his limbs. He has still flying, shooting pains down them, principally confined to the back, often beginning in the heel and shooting upwards. He complains of the sensation of a tight gutta-percha band encircling his thighs a little above the knee, and contracting or expanding with the pains. These pains come on in paroxysms, at all hours of the day or night, perhaps more frequently in the afternoon. When they occur at night, he is obliged to get out of bed, because he cannot bear the contact of the bedclothes. He has no deep-seated, boring pains. During the paroxysms his legs are jerked and spasmodically stretched out, as if they were being galvanized. Within the last three weeks the fits have recurred less frequently, and when they have come on they have affected limited patches only. The sensibility to pricking and to temperature is perfect. Tactile sensibility is very obtuse, but less so on the inner than on the outer surface of the limbs. An interval of five seconds and more, elapses, before he perceives an impression of mere contact. His voluntary movements resemble those of persons affected with chorea. His legs are thrown wildly forward and suddenly extended with a jerk, and brought down with force on the ground from the heel to the toes. His gait is unsteady, but not shuffling. He is obliged to use a stick when walking, but he does not look at his legs. In fact, looking at his feet makes him giddy, and renders his walk more unsteady still. When his eyes are closed he cannot keep his balance when standing, although his feet be wide apart.

The strength of his muscles is considerable, and he can successfully resist all attempts at bending or stretching his limbs against his will, even when his eyes are closed. When he stands without resting on his stick he has curious sensations, and an inclination to jump upwards; in fact, he feels as if he stood on a springing board. When he lies down and closes his eyes, he has only a confused notion of the position of his limbs after they have been shifted about, until he begins to contract his muscles. He can, at will, however, slowly, or with different degrees of rapidity, flex, extend, or place his limbs in any given position, without looking at them. The electric contractility of his muscles is perfect, but their sensibility to electric currents is considerably below par.

The *upper extremities* have lately become affected with shooting pains similar to those in the lower limbs, and chiefly felt along the inner border of the forearm, and extending to the little finger. The *left* arm is worse than the *right*. There is a sensation of numbness in the tips of the fingers, but tactile sensibility seems normal, and there is no appreciable retardation in the conduction of tactile impressions. Pricking and differences of temperature are immediately perceived. There is some awkwardness in the movements of the fingers, but the patient can undo the button of his shirt collar, which he does not see. He can grasp firmly with either hand.

June 24th.—The paroxysms of shooting pains have recurred more frequently lately, and have been very severe in the legs. Whilst they last, the legs are convulsively agitated, and the slightest touch makes him shrink; but there is no change in either pupil; the right contracted pupil does not dilate in the least.

September 5th.—Since the last note, the patient has improved considerably. He has gained both flesh and strength, and he is in very good spirits. He can walk across the whole length of the ward without using a stick, and, although he still staggers and totters much, there is, however, less inco-ordination than previously. His tactile sensibility has improved; he feels a mere touch, accurately and at once, and he can tell, without looking at them, the position of his limbs, however much they may be shifted. The paralysis of the *left* upper eyelid, which had been growing less and less since the beginning of July, has completely disappeared for the last fortnight. The *left* pupil is still dilated, and the *right* contracted to the same degree. The diplopia persists also; objects seen with the left eye are brilliantly illuminated and look too white; those seen with the right eye look dark. There never is, at any time, the slightest attempt at erection. He still has frequent paroxysms of pain, affecting the upper more frequently than

the lower extremities. The perspiration of the left half of the forehead during the paroxysms of pain is still strikingly marked.

Case 5.—W. E. A——, aged 32, formerly a printer, of late years employed in the shoe trade. He is married, and has two children, the younger of whom is six years old. He is thin, middle-sized, and pale, and has fair hair and whiskers. There is no history of nervous diseases of any kind in his family, as far as he is aware of. About fifteen or sixteen years ago, he had simple chancres, followed some time after by an eruption of roseola, for which he was carefully treated, and which never recurred subsequently. He has had nocturnal seminal emissions as far back as he can recollect, occurring on two successive nights, and generally at intervals of a week. He has all his life also suffered from painful and laborious digestion. With these exceptions his health has, on the whole, been good.

His present ailment dates from eight years ago or thereabouts (1856 or 1857). The first symptom which attracted his attention was an acute throbbing pain in both his lower limbs, so much like the pain of toothache that he used to call it *toothache in his legs*. This pain came on in paroxysms, at all hours indifferently, but more especially about the early morning, even whilst lying quietly in bed. It did not always attack the same portion of the limb, but began in any part of it, and from there shot off to the rest. No change occurred in the colour or the temperature of the parts affected. It seemed very superficial, and was greatly exaggerated and intensified by the lightest touch—by the mere contact of the bed-clothes: deep pressure, on the contrary, did not affect it, neither relieving nor increasing it. No embrocations or external applications of any kind that he tried ever had any influence in checking or alleviating it, but after lasting a variable number of hours, it disappeared spontaneously.

Until two years ago (1862) no other symptom was noticed by him. At that time the ring and little fingers of his *left* hand began to feel numb, and this sensation of numbness gradually crept upwards as far as the elbow, but no higher. From that time also he, by degrees, lost more and more the command over the movements of his left fingers; and he found that unless he kept looking at any object he might chance to carry in his left hand, he was apt to drop it. Within the last few months, the fingers of his right hand have in their turn felt numb, but he has still perfect command over their movements.

Simultaneously with the sensation of numbness and the gradual loss of co-ordination in the left hand, in 1862 his lower limbs became affected with *weakness*, as he thought. He got easily tired when walking, was constantly tripping, and had a tendency to lose his balance; he rolled very much, and staggered like a drunken man,—so much so, that his wife's attention was particularly attracted to it. Weakness of the bladder also set in, for he could not retain his urine as soon as he felt the want to pass it. He did not notice that he was obliged to watch his legs constantly when walking or standing, and that he could not walk in the dark, until December, 1863, shortly before Christmas, when, feeling feverish and thirsty one night, he got out of bed in the dark, in order to get some water to drink. He was greatly surprised and distressed, however, on finding that his legs nearly gave way under him, and that he was unable to move about unless supporting himself by taking hold of the various pieces of furniture in the room. Before then he had regularly walked to his place of business, a distance of about a mile from his house, every morning, but he now found the exertion too great for him, and feeling that he was getting gradually weaker, he gave up his employment. Numbness of the soles of his feet, at first trifling, became, from this time also, more and more highly marked; he had the sensation of cushions placed between his feet and the ground; and his gait gradually assumed its present peculiar tottering character. His sexual power became by degrees less and less, and within the last three months it has been totally extinct.

His sight has never been affected, and he has had no squint. Of late, he has had pains, similar to those in the legs, affecting the trunk of the body and both his arms, more particularly the *left*.

He became an in-patient in the beginning of June, 1864, when the following notes were taken :—

Intellect unaffected ; no pain in the head ; spirits very low and depressed ; manner, that of a highly nervous individual. His hearing is good ; his sight also. Both pupils are contracted, and equally so ; the globe of the eye is not injected, and the patient has no sense of increased temperature in the eye. There is no strabismus. His articulation is perfect. Circulation feeble ; pulse 66, weak, and easily compressible. Appetite good ; his bowels have a tendency to be confined, but they have always been so all his life. The *bladder* is weak : the urine comes away in a small stream, not a full arched jet, and he is obliged to strain whilst passing it : there is no stricture of the urethra. He has no involuntary dribbling, no nocturnal incontinence, but he cannot retain his urine for any time after he feels the want to pass it. All power of erection is completely lost, and since the last fortnight he occasionally has seminal losses unattended with sensations. There is no spontaneous pain along the spinal column, and none is elicited by quick movement of the trunk forwards and backwards, by hard knocking of the spinous processes of the vertebræ, &c.

Both *upper extremities* look equally well nourished. The motor power of the *right* arm is not in the least degree impaired, but the patient complains of a numbness of the tips of the fingers : he can, however, tell at once when and where he is touched, scratched, or pricked. In the *left* hand, the numbness is not confined to the tips of the fingers, but extends over the whole hand, both the dorsum and the palm, and reaches along the inner half of the forearm as high up as the elbow. He can, nevertheless, distinguish at once the exact spot where he is touched when his eyes are closed. This *left* arm feels weaker, but is not so in reality, for he can grasp firmly with his left hand, and as well as with the right, although he cannot regulate and guide its movements. If asked to pick up a small object, his fingers go by the side of it in a sort of jerking convulsive manner, as in chorea, and it is only with difficulty that he can take the object up between his fingers. If, whilst his eyes are closed, a small box two inches square, be placed between his hands, he either lets it drop altogether after a minute or so, or turns it upside down, without being conscious of the act. He cannot tell the weight of objects placed in his left hand, unless the difference in weight be considerable. He occasionally suffers from shooting pains in both arms, particularly marked in the left, and similar to those in the legs, only less intense.

His *lower extremities* look well nourished, and are well developed for a man of his size and stature : they have not the look of palsied limbs. To the touch they are of the same temperature as the rest of the body, nor do they feel cold subjectively. When in bed, the patient does not know their position under the bed-clothes. When his eyes are closed, if he be asked to execute movements with his lower extremities, such as lifting them off the bed, flexing or extending them, he does so instantly, but with great irregularity and disorder, to a much greater extent than he is aware of, for he cannot regulate the degree and completeness of the movements. When he looks at his legs, he can perform the same movements with less irregularity, but still without precision. When his leg is fully extended, it cannot be bent against his will by another person, thus showing the power of his muscles. If he is asked to rise and stand, the efforts he makes are striking, and take one by surprise. He cannot get off his chair without laying hold of a neighbouring piece of furniture. Mere standing becomes impossible after a short time, unless he leans on a stick. If his eyes be shut, this helplessness is still more manifest, for he oscillates, loses his balance, and soon threatens to fall down unless propped up. His walk has the same characters as those described in the preceding cases. He can only walk very

short distances, and is obliged to halt frequently. He keeps looking at his legs when he walks, but the choreiform character of his walk is not thereby diminished.

The tactile sensibility of his feet and legs is considerably obtuse, and the impressions are slowly conducted to the sensorium. Pricking and differences of temperature are easily felt and distinguished. Electro-muscular sensibility is considerably below par; powerful electric currents, which make the muscles contract most energetically, cause scarcely any pain. He is still liable to the paroxysms of shooting pains described in his previous history.

Sept. 10th.—For the last five or six weeks the patient has been obliged to use crutches. The incoordination of his movements is still more marked than previously, and he cannot move a single step without his crutches. There is not the slightest wasting of the legs; the impairment of tactile sensibility in them is greater in degree, but there is the same acute perception of painful impressions as in health. The numbness of his hands has also increased considerably, and the movements of his *right* hand are now beginning to have the ataxic character; those of the left hand are very remarkably choreiform.

Both pupils are contracted to the size of pin's points, but there is no impairment of vision. The paroxysms of shooting pains are less frequent and less intense: they affect the four limbs, but chiefly the lower, and are not felt in the trunk.

Case 6.—G. B——, a cabman, aged 32, unmarried, became an out-patient, under Dr. Radcliffe's care, at the National Hospital for the Paralysed and Epileptic, in the beginning of June, 1864. He is a middle-sized, thin, pale man, with prematurely grey hair (his hair began to turn grey when he was only eighteen years old). He is an habitual hard drinker, and used to get drunk two or three times a week until lately. He had chancres and suppurating buboes eight or nine years ago, but no secondary symptoms since. From the nature of his occupation, he has been much exposed to wet. Upon the whole, his previous health has been good. He has never had puffiness of the eyelids on waking in the morning, or swelling of the ankles.

He dates his present complaint from the beginning or middle of March, 1864. He had some difficulty in walking, and staggered and rolled a little, particularly on first getting off his box. As this difficulty was very slightly marked, and disappeared spontaneously after he had walked about for a little while, he paid no attention to it. On the 1st of April, however, when getting down from his cab about midnight, after a long day's work, he felt his legs give way under him, and he staggered so much as he walked on to a public-house to light his pipe, that he was accosted by a policeman, who, believing him to be drunk, told him that he had had enough, and had better go home. He had no pain in any part of his spine at the time. The next day he felt worse; his legs were much weaker, he staggered more, and he complained of giddiness. He tried to drive his cab as usual, however, in spite of these feelings, but was obliged to give up the attempt. His arms were strangely affected, for although he could drive very well, he could not use his whip, because it seemed to him *as if his arm would go with his whip*. He felt extremely cold all over, and complained of tingling in the tips of all his fingers and toes, as well as of numbness and sense of weight in all his limbs. There was no head-symptom, beyond mere giddiness when walking. He had no headache, no confusion of ideas, no drowsiness or sleepiness, no affection of the sight, no nausea or vomiting. He consulted a medical man, who gave him some medicine, and for about a fortnight the weakness of his limbs diminished, and he tottered less in walking. Since then, however, that is for the last five weeks, he has been as bad as he is now.

Present state, June 4, 1864.—Intellect unaffected; spirits, however, very depressed at times. Within the last two or three weeks he has had occasionally, but not constantly, a sensation of weight and a dull pain exactly limited to the

back of his head. When he walks he feels very giddy, but when he sits down quietly, he has no such sensation. His hearing is good. Within the last two or three days he has had *double vision*, but he sees perfectly well with either eye alone. When he uses both eyes, he sees an object *single* at a distance of about two feet from him; but as the object is moved farther away, he sees it at first as through a mist; and at a distance of two feet and a half, he begins to see two distinct objects instead of one. He has no ptosis, no strabismus, but the *left* pupil is nearly double the size of the *right*. His face is symmetrical, and both halves move equally well. The sensibility of both cheeks is equally good. There is no tingling of the lips. The tongue is protruded well, without tremulousness; its apex does not deviate, and its surface is clean. The voice is unaffected, and the articulation distinct. Deglutition is normal; appetite very good; digestion easy; he never has nausea or actual sickness. Bowels regular; bladder unaffected; no difficulty in passing his urine; no dribbling; urine free from albumen. No pain is elicited by knocking or making hard pressure on the spinous processes of the vertebræ. No spontaneous pain exists, except after he has been walking for a little while, when he has a sensation of dragging, from between the shoulders downwards, along the spine.

Upper extremities.—He has a sensation of extreme cold in both arms, more especially the right, as if he had rested his bare arms on a cold marble slab. To the touch, however, the arms do not feel colder than the rest of the body. He complains of tingling and pins and needles in the tips of his fingers, and of numbness of both upper extremities. Tactile sense is not diminished, however, for when his eyes are closed, he can tell distinctly and at once where he is touched; when tried with the compasses, he detects the two points within normal limits. The sensibility to pain and to differences of temperature is normal. His muscles contract fairly under the influence of galvanism, and he is conscious of their doing so. He can grasp firmly, although he complains of a certain diminution of power in his arms. This is more imaginary than real, and arises from his ascribing to weakness a deficiency of coordinating power, for he always spills his tea whenever he raises his cup to his mouth.

Lower extremities.—He has the same sensations of cold and numbness in these limbs as in the arms; he complains also of tingling in the toes. The various kinds of sensibility are normal; and when he walks, he has no sensation of a cushion or pad intervening between his feet and the ground. He cannot walk without the help of a stick, and even then his gait is peculiarly tottering. His legs are thrown about as if he could not measure the strength required for moving them, and his heel comes down first. He cannot stand with his eyes closed, for he quickly oscillates and loses his balance. His legs look well nourished, and have not wasted in the least since he has been ill. They possess considerable power, and cannot be bent against his will. On tickling the soles of his feet, no reflex contractions are excited in the limbs. At night, however, he has involuntary jerks and spasms of the legs. When he first begins to walk, he staggers and reels more than he does after he has been walking for some little time. When walking, he has to stop short before he can turn round, otherwise he is apt to lose his balance.

At the end of June, his diplopia had disappeared, and he no longer felt giddy when he turned his head round over one shoulder. He went on in the same way without any great improvement until October, when for the space of a week he had great difficulty in passing his urine. Six months afterwards, he began to notice a failure of his sexual power, which until then had been unimpaired, and he had occasional nocturnal emissions, unaccompanied by any sensation. Dilatation of the left pupil again made its appearance, and with it the diplopia. He also complained of a fresh symptom, namely, paroxysms of frightful pain, chiefly affecting the knee-joints, and the left more than the right, which he compares to the sensation of a wire being quickly thrust through his knee. When he was last seen at the end of July, 1865, his gait was very characteristic of locomotor

ataxy. He still complained of the paroxysms of pain in the lower extremities, and of aching pains in both wrists. The movements of his hands were markedly ataxic, for he could not touch the tip of his nose at once, when asked to do so with his eyes closed. His left pupil was still larger than the right, and, although his sight was not then misty, and was good when he used only one eye at a time, he saw double when he used both eyes together. There was no stra bismus. Within the last few weeks, he had noticed that he could not tell the position of his limbs in bed, under the bedclothes. He still complained of numbness of the legs, and yet, strangely enough, when tactile sensibility was tested with the æsthesiometer, it was found unimpaired, for he could feel the two points of the instrument at a distance of only two inches, when applied in front of the legs. He has a sensation of tightness over the abdomen, as if it were compressed by tight clothes.

Case 7.—J. S——, aged 43, a bootmaker, married, without children, has six sisters living in good health; his father and mother died at the advanced age of 80. He is no smoker, has not been a hard drinker, and has never committed sexual excess of any kind. He is a medium-sized, apparently well-nourished individual, of very pale complexion. About four years ago, he began to complain of a sensation of cold in the occipital region on lying down. He could not see well with his left eye, and was troubled with black spots and occasionally with globes of fire in the field of vision of that eye. His general health, which had previously been very good, failed also about the same time, and he became subject to peculiar nervousness and to fits of despondency, and occasionally to a tendency to fainting. For nearly three years his health was in that unsatisfactory condition, when in August or September, 1863, without his being able to assign any cause for it, he became affected with sharp, shooting pains and twitches in both lower extremities. These pains chiefly attacked the knee and ankle-joints; they came on in paroxysms, and went off with extreme rapidity: he compares them to the pain felt in tic-douloureux. They occurred in the daytime perhaps more frequently than at night, and were not apparently influenced at that time by season or change of weather. He had no pain in the spine, and he asserts most positively that there was nothing amiss with the motor power of his legs, for he could run and jump as well as ever, and he has always been an active man. He noticed, however, some diminution in the expelling power of the bladder, for he was a longer time passing his urine than before, and the jet was not arched as in health. About Christmas, 1863, another symptom made its appearance, besides those mentioned above, namely, occasional numbness of the ulnar half of both forearms and hands, and of the ring and little fingers, as if he had been lying on that part; rubbing removed the feeling. He did not give up his usual occupation until the end of March, 1864, and that was in consequence of a severe attack of pain which affected the left half of his chest—probably intercostal neuralgia. On this pain disappearing after a week, he resumed work, but a fortnight afterwards he was taken ill one night, during sleep, with sensations of faintness coming over him. In the morning he found that his *left upper eyelid* had dropped a little, and two or three days after this, he had double vision, the second object appearing at a height of between twenty or thirty yards above the other. About the same period he began to complain that his legs *felt heavy*, and on trying one day to run, he found that he could not manage it. He could feel the ground perfectly well at that time, and he affirms that he could walk in the dark. In July, the *heaviness* of the legs seemed to go away and to be replaced by weakness: he tottered very much, and shook on his legs when walking; but he did not feel giddy. The paroxysms of pain came on very frequently, more so than before, and attacked the whole lower limbs, and not the knee and ankle-joints only. The ptosis and diplopia disappeared about the end of July, and have not since returned.

When he came under my observation for the first time, in December, 1864, I

noted down the following points:—No head symptoms; memory good. Sight and hearing normal; pupils equal and natural. No pain in spine, spontaneous, or on knocking of spinous processes of vertebræ. No sensation of a tight cord round the waist. Bladder still weak in expulsive power. Sexual aptitude very much impaired, and has been so for months. Has had seminal emissions once or twice only, and that within the last fortnight. *Upper limbs:* Their movements are not marked by incoordination or irregularity. Occasionally, but rarely, they are the seat of shooting pains, coming on in paroxysms, and chiefly felt along the ulnar side of the fore-arms.

The *lower limbs* look well nourished, and do not feel cold to the touch, neither has the patient any subjective sensation of cold in them. Their sensibility to pain and to differences of temperature is perfect. Tactile sense, tested with the æsthesiometer, is also found to be normal, and there is no retardation in the conduction of impressions to the sensorium. He does not, however, know the position of his limbs after they have been shifted about when his eyes are closed, until he begins to contract the muscles of the limbs. His electro-muscular sensibility and contractility are unimpaired. When the sole of his foot is tickled, he has only a sensation of mere contact, and there is no reflex drawing up of the corresponding limb. When he is sitting or lying down, he can flex or extend his legs with different degrees of rapidity, even with his eyes closed, although in the latter case the movements are irregular and disorderly. I cannot succeed in bending or stretching out his leg against his will; thus showing that he possesses a considerable amount of power. When he is asked to stand, however, the difficulty which he has in getting up from his chair contrasts strangely with the amount of power which he displays when sitting. He is obliged to take hold of some neighbouring object in order to rise, and his legs shake under him all the while. When once on his legs he no longer needs any point of support, but the muscles in front of his leg and foot are seen to contract spasmodically, and he looks as if he had a tendency to jump upwards. When he is asked to shut his eyes, he immediately loses his balance, oscillates, and would fall down, unless he were supported. His gait is eminently characteristic of ataxy: there is the usual spasmodic extension of the leg before it has completed its oscillation, and the heavy bringing down of the heel, the inability of controlling and regulating the degree of muscular contraction, and of executing certain movements, such as turning round sharply, &c.

In the beginning of 1865, he was, for a few weeks, admitted an in-patient, and whilst in the hospital he was very frequently attacked with sharp, shooting pains in the legs and arms, but chiefly the legs. These paroxysms were more frequent and severe on cold and damp days, and the incoordination of his movements was always more marked after them. The movements of the arms gradually became irregular also, and by the end of April, 1865, since which time he has been lost sight of, the ataxic character of the movements of the upper limbs was well marked. The diplopia had not returned, and the sight was good.

Case 8.—S. D——, aged 38, married, a mantle-maker, was admitted into the National Hospital for the Paralysed and Epileptic, under Dr. Radcliffe's care, on May 1, 1865. She is tall, well nourished, with the aspect of health, although of a sallow complexion. Her previous health has been very good, notwithstanding her getting frequently wet on her way to the City in the morning, and her having to sit in her damp clothes all day. She has lived well, and ascribes her present ailment to the cares and troubles which she has had since her husband deserted her. She has always menstruated regularly. Her present complaint began about Christmas, 1860, when she became affected with pain in both legs, coming on suddenly, in paroxysms, and going off as suddenly, of a sharp, shooting character, and often attended with drawing up of the legs. On several occasions, when seized with this pain whilst walking, she fell down in the

streets, from her legs giving way under her. Troublesome numbness of her right leg and foot set in about the same time. For the space of eighteen months these were the only symptoms which she noticed. The pain came on in paroxysms for two or three days in succession, several times in the day, and then left her for a week. It never attacked the whole of the limbs at the same time, but only limited portions of them, and never the same spots in two succeeding paroxysms. It was always accompanied by involuntary drawing up and spasmodic jerks and starts of the limbs.

At the end of eighteen months, the numbness extended from the right knee upwards to the hip, and to a less degree affected the whole of the left limb also. When she walked she was obliged to look at her right foot, because it seemed to her as if she were treading on sponges on that side, but not on the left. Whenever she went up a staircase, also, her right foot started up involuntarily the height of two stairs at a time, so that she was always in danger of falling down. By Christmas, 1863, the left leg had become as bad as the right, with respect to the numbness and the inability to control and guide its movements. From that time she was obliged to use a stick when walking, and she was no longer able to walk in the dark. In November, 1864, she had to give up walking by herself, even with the help of a stick, because her feet got entangled in one another, and she was in constant danger of falling down. For the same reason she could not go about resting on two sticks or on crutches; and yet strangely enough, she has always been able, when leaning on a friend's arm, to walk a short distance. In the beginning of 1865 she complained of numbness of the inner half of the right hand and fore-arm, followed, two months afterwards, by paroxysmal pain of the same character as that which attacked the legs. The pain was felt, also, along the inner surface of the right arm, but without being accompanied by numbness. About the same time as her right arm became implicated, her sight began to fail. Thus, after she had been at work for some time, her sight would grow misty at first, and then she would have double vision, and after a short time again she would become entirely blind. On resting her eyes for a little while, however, her sight gradually returned; but these attacks recurred so frequently that she became very anxious lest she should get totally blind. She affirms that she never squinted. She never had frontal or temporal headache before or during the fits of temporary blindness. From the time her sight became affected, the paroxysms of pain to which the lower limbs were liable grew much worse as to frequency and intensity.

During her residence in the hospital I carefully examined her on several occasions, and took the following notes of her case:—

Intellect unaffected, memory excellent, but spirits very depressed; shakes and trembles all over when addressed. She is not subject to headache, and has no sensation of weight in the occipital region. Hearing, smell, and taste are perfect; articulation clear and distinct; face symmetrical; no twitchings of facial muscles. Tongue protruded well in a straight line, without tremulousness. No dysphagia. Respiration normal; deep and forced breathing regular. Circulation very feeble; pulse extremely weak and compressible. Heart sounds normal but feeble. Appetite capricious; bowels regular. No sensation of tightness across the abdomen. Bladder unaffected; micturition normal; no dysuria; no incontinence. Menstruation regular, and of normal quantity. Her sight is very weak: she is not able to read but a few minutes at a time, and intermittently, as it were, for the lines run into one another; she sees double, and then becomes totally blind for a short time. Her conjunctivæ are not injected; her pupils are equal but large. She moves both eyes well upwards, downwards, outwards, and inwards. She can see better with the right eye alone than with the left. The paroxysms of pain do not seem to affect her sight in any way, neither making it worse nor improving it, and do not alter the size of the pupils.

Upper extremities.—On the *left* there is no numbness and no pain; the movements performed with that limb are not wanting in precision, regularity, or

energy, even when the patient's eyes are shut. On the *right* side, on the contrary, there is frequently present a sensation of numbness in the inner half of the hand, fore-arm, and arm ; and paroxysms of pain frequently attack that limb several times in the day. The movements performed on that side are extremely deficient in precision and regularity, and are somewhat jerking in character. The patient is unable to carry a cup of tea to her mouth with that hand, without spilling some of the contents. That there is no real loss of muscular power is clearly shown by the firmness with which she can grasp and squeeze my hand. The pain caused by the pricking of a pin or by pinching is normally felt on the left side, whereas along the inner half of the right limb it is much less acutely felt. Weights placed in the left hand are correctly appreciated, but several half-crowns placed in the right hand feel less heavy than one half-crown alone in the left hand. Differences of temperature are equally estimated on both sides ; but as regards the acuteness of touch, there exists a remarkable difference between the two hands. Thus, on the *left* side, the two points of the æsthesiometer are distinctly felt in the anterior half of the ungual phalanx, when separated by an interval of two-tenths of an inch, and in the posterior half of the same phalanx when three-tenths of an inch apart. On the right side, on the contrary, the two points are felt only when three-tenths of an inch apart in the anterior half, and four-tenths of an inch in the posterior half of the ungual phalanx. Similar differences exist between the measurements of the rest of the hand and fore-arm on both sides.

The *lower extremities* look well nourished and feel firm to the touch. When the patient is sitting or lying down, she can flex or extend her legs with force and rapidity, and I cannot succeed in bending them against her will on using moderate force. On asking her to stand up, however, the difficulty which she has in getting up strangely contrasts with the amount of force which she displays when sitting down. She is obliged to take hold of the chair or bed, and even then she is extremely unsteady as she gets up. She cannot stand by herself except with her feet wide apart, whilst the muscles over the dorsum of her foot and in front of her leg are seen to be thrown into spasmodic contractions. She is soon obliged to sit down, because the act of standing fatigues her very much, as it compels her to keep her will on the stretch, as it were, in order to maintain her equilibrium. When she walks, her gait, even when she leans on a friend's arm, is extremely unsteady. She takes only very short, quick steps, throwing her legs forwards with a jerk, and bringing her foot down with a heavy tramp on the heel. As usual with all ataxic patients, she is obliged to stop abruptly short before turning round. On account of her dress she cannot see her legs, so that sight cannot help her to guide their movements ; yet, if she be asked to shut her eyes, she cannot stand for a single moment; thus showing the influence of light apart from visual sense on the coordination of movements. She cannot appreciate the resistance of the floor on which she walks, and has a sensation as if she were treading on sponges. In bed, at night, she has not the least notion of the position of her limbs, and if one of her legs be placed over the other, she cannot move it unless she can see it. Tactile sense is considerably impaired in the soles of the feet and in the legs, and less so in the thighs. Painful impressions themselves are less acutely felt in the feet and legs than elsewhere ; but differences of temperature are correctly appreciated on both sides. She is liable to frequent attacks of sharp, shooting pain in her legs, but she is better in that respect since admission.

She left the hospital in the beginning of August, 1865, in every respect in an improved condition. Her gait was less unsteady, and she could even manage to walk across the ward without leaning on a friend's arm or even using a stick. Her sight had improved considerably, and she could read for fifteen and twenty minutes at a time. Two months afterwards, however, she had gone back to her former condition, probably from being no longer placed in the same favourable hygienic conditions as during her stay in the hospital.

The first inference, which may be drawn from the above series of cases, is the comparative immunity of the female sex from progressive locomotor ataxy; for of the eight patients whose history has been related, one alone is a female. The same circumstance has been already noted by other observers, and Professor Trousseau states that he has only met with three cases of this disease in females, although, at the time when he made that statement, he had already seen more than fifty instances of it. Dr. Duchenne (de Boulogne), also, had seen progressive locomotor ataxy in females four times only. On the other hand, Dr. Topinard,[1] in his exhaustive monograph on this disease, gives 33 females *versus* 81 males, out of 114 cases collected by himself, whilst of 70 cases brought together by Eisenmann,[2] in 20 only were the patients females. This relative protection of the female sex has not been noticed in the case of progressive locomotor ataxy alone, but seems to obtain in all forms of disease of the spinal cord, as would appear from a table published by Dr. Brown-Séquard,[3] showing that of 177 cases of paraplegia, 128 occurred in men and only 49 in women.

The age of the patients in the above group of cases ranged between thirty and forty, thus pointing to adult age as the one most prone to the disease. Professor Friedreich, of Heidelberg, is the only one as yet, as far as I am aware, who has published cases of progressive locomotor ataxy occurring in individuals who had not yet reached adult age, three of his patients being aged respectively fifteen, sixteen, and eighteen.[4]

There is no special cause to which may be traced the origin of the disease in the above cases; for if, in one half of them, the patients were, from the nature of their occupation, much exposed to wet, in the other half, on the contrary, no such exposure was ever possible, at least for any prolonged period. Some of the patients spoke of grief and mental trouble as having brought on their complaint, and in many instances of progressive locomotor ataxy, recorded by various authors, this cause is very much dwelt upon. Yet, in what manner, and through what channel, any deep emotions or depressing passions, however prolonged, can act in bringing on this peculiar complaint, it is difficult to understand. Sexual excess has by some been regarded as the chief cause of the evil, but in only one of these eight cases is there mention made of

[1] Topinard: "De l'Ataxie locomotrice," &c. Paris, 1864, p. 360.

[2] Eisenmann: "Die Bewegungs Ataxie." Wien, 1863.

[3] "Lectures on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities," by C. E. Brown-Séquard, M.D., p. 100.

[4] Friedreich: "Ueber degenerative Atrophie der spinalen Hinterstränge" (Virchow's Archiv für pathologische Anatomie, Nos. 26 und 27, 1863). See a translation of the same in "Archives générales de Médecine." Paris, 1863.

long antecedent spermatorrhœa, probably the result of excessive onanism. In all the rest no sexual excess of any kind is admitted, and, in fact, the class of society to which the patients belonged is not one peculiarly prone to this kind of vice. The abuse of alcoholic drinks, again, cannot be assumed as a cause, for only one of the eight patients acknowledged to have been an habitual hard drinker. With regard to syphilis, in only one case had there been anything like it, and even then the disease had assumed its mildest form, that of roseola, from which the patient had suffered fifteen or sixteen years before any symptom of progressive locomotor ataxy made its appearance. In two other cases simple chancres had been contracted at one time, but they had never been followed by secondary manifestations. As to the relation between rheumatism and ataxy in these cases, none could be traced. The patients, it is true, complained of what they called *rheumatic* pains; but they thereby meant to designate the peculiar erratic, flying pains characteristic of locomotor ataxy; but in no instance had there been a genuine attack of articular rheumatism.

The question concerning hereditary predisposition to the disease, either directly, or indirectly through a transformation of an inherited tendency to the development of nervous disorders, as so ably pointed out by Professor Trousseau, is answered in the negative by the history of the above cases. In none was there any mention made of paralysis, epilepsy, or other nervous affection having existed in any member of the family. That such predisposition, however, does sometimes occur, the cases mentioned by Professor Trousseau clearly demonstrate, as well as those published by Friedreich, which include two individuals of one family and four of another. But a still more remarkable instance is that related by Dr. Marius Carré, of a family, eighteen members of which became ataxic in turn; namely, the grandmother, the mother, all the relatives of the latter, eight in number, seven children, and one cousin.[1]

The slow and insidious manner in which the disease usually sets in, is well exemplified in seven of the above series of cases, and in one only do we meet with the sudden and quasi-explosive manifestation of symptoms, of which Dr. Topinard relates a certain number of instances. The patient in that case (No. 6) most positively affirmed that he was apparently enjoying very good health at the time, when he was suddenly seized with numbness of the arms and legs, and with inability to guide and regulate the movements of those limbs. In all the other cases we find the disease creeping in, imperceptibly as it were, and not

[1] Marius Carré": De l'Ataxie locomotrice progressive." Thèse de Paris, 1863.

assuming its special physiognomy until after the lapse of many months and even years.

A review of the symptoms on the whole, and of the order in which they manifested themselves in the above cases, admits of dividing the course of the disease into two periods or stages. The first of these would correspond with Professor Trousseau's premonitory stage, namely that which includes the attacks of paroxysmal intermittent pain, the spermatorrhœa or impotence, the weakness of bladder, the paralysis of various motor nerves of the eyeball, and the disorders of vision. It is hardly possible, however, to regard these merely in the light of premonitory symptoms, because they form part and parcel of the fully developed disease; and although in the majority of instances they precede, yet they sometimes only appear after, or simultaneously with, the motor incoordination, which, together with the loss of sensibility to touch, and in some cases to pain, characterizes the second stage.

In six out of our eight cases, *pain* was the first symptom which attracted the patient's attention; and, in all of them, it showed itself in the course of the disease, becoming, after a time, a prominent symptom, in fact, the most troublesome and harassing to the patient, wearing his strength out, and depriving him of rest. This symptom, however, does not seem to be invariably present in all cases of progressive locomotor ataxy, for Dr. Topinard [1] states that it was absent in 22 out of 104 cases, although he justly adds that it was in all probability present at some time or other, and was simply omitted from the extreme concision with which these twenty-two cases were reported. When it first made its appearance in the above cases, this pain did not assume its special and distinctive characters, but was in general spoken of as a dull aching, similar to that ascribed to muscular rheumatism. It usually affected both lower limbs simultaneously, but sometimes one of them only, or at least one limb with considerably more intensity than the other. A circumstance worth noticing is, that the limb which was first seized with pain, was also the first to become affected with incoordination. The same remark applies to the upper extremities, and in many of the above cases we find one arm the seat of pain and of ataxy, to the exclusion of the other.

After having lasted for a variable time, this pain assumed characters which may be said to be almost typical of the disease. All the patients spoke of its shooting, flying, erratic nature, of its sudden appearance, and its as unaccountably sudden disappearance, and of its recurrence in numberless paroxysms. Two spoke of it as

[1] Topinard: *loc. cit.*, p. 150.

toothache in the legs; another compared it to the pain of *tic douloureux;* and whenever the paroxysm was very severe, they all described it as similar to the pain produced by the passage of a powerful electric current through a limb. In one case alone (No. 6) was it spoken of as being of a boring, perforating character (*douleurs térébrantes* of Duchenne), the patient comparing it to the sensation that would be produced by the rapid thrusting of a wire through his knee-joint. As a rule, the shooting, lightning-like pain was more frequent (*douleurs fulgurantes* of Duchenne, *blitzenische* of Romberg). During these paroxysms, the affected limb was agitated involuntarily, jumped and startedas if a galvanic discharge was being transmitted through it, whilst no change occurred in its colour and temperature. The lightest touch, mere contact of the bedclothes, I have found, in most instances, to exaggerate and intensify the pain, whereas in a few only did firm constriction of, and deep pressure over, the part relieve it a little. In some no relief was thus afforded, although no increase of pain followed on deep pressure. As a rule, however, the patients were very averse to trying the effects of pressure, and when the pain was very severe, they nervously dreaded the approach of any one. Changes of weather from dryness to dampness, and from a high to a low temperature, generally brought the pain on; but the reverse was sometimes true, and I have known patients attacked with the most frightful paroxysms of pain on the temperature rising suddenly in spring, a period of the year during which ataxic patients are well known to improve remarkably.

When the lower extremities were the seat of those peculiar pains, any portion of them was liable to be at any moment attacked. But this was not the case with the upper extremities, for when they were involved in the disease, the pain was always limited to the inner half of the fore-arm and palm of the hand, and the ring and little fingers (to the parts, in fact, supplied by the ulnar nerve). Dr. Duchenne[1] (de Boulogne) mentions one case, however, in which the outer portion of the hand—that supplied by the median nerve—was affected. In one of the above cases (No. 8) the pain was also felt along the inner aspect of the arm.

Besides these paroxysms of shooting pain, the patients often complained of a sense of constriction in various parts of the body, the trunk as well as the extremities. In case 1, there was at one time a sensation, as if the base of the chest were constricted by a broad bandage which impeded respiration; in cases 3 and 4, the sensation of constriction of the abdominal walls, as if pressed down by tight clothes, was at one period con-

[1] Duchenne (de Boulogne): "De l'électrisation localisée," p. 557.

siderably marked. The wrists, ankles, and knees often also felt as if they were encircled by a tight band, and one patient (case 4) even added that during the paroxysms of pain, these compressing bands seemed to be alternately tightened and relaxed, thus giving him the idea of india-rubber bands.

In the history of six out of our seven male patients, impairment of the sexual power is mentioned. One man alone (case 3) declared that he had noticed no difference in that regard, although he was in an advanced stage of the disease, which had already involved one of his upper extremities. In one case (No. 5) spermatorrhœa had existed for a considerable number of years previous to the development of locomotor ataxy, and it had ended at last in complete impotence by the time the patient was admitted into the hospital. In spite of his spermatorrhœa, however, he had had two children. W. R——, also (case 1), had had a child in the fourth year of the disease, although suffering at the time from marked diminution of sexual power. As to the other four male patients, two were absolutely impotent when they came under my observation, and the remaining two admitted a considerable diminution of the sexual appetite and vigour, with occasional seminal emissions, generally unaccompanied by sensations.

In all the male patients, without exception, the bladder was affected: the woman, S. D—— (case 8) alone escaped this complication. The functions of the organ were interfered with in a slow and gradual manner. Thus, the patients first noticed that they were a longer time than usual in passing their urine, that the jet did not describe the same arch as in health, or escape with the same force; these facts showing that the fundus vesicæ was losing its energy. Next there came on inability to retain the urine long, frequent calls to pass it, and that, immediately on feeling the want, under pain of its escaping; and lastly, actual incontinence. In some cases this weakness of the bladder was spoken of as a constant, persistent phenomenon; in others it was intermittent, and recurred chiefly after exertion or in connection with paroxysmal pain attacking the perinæum. To these functional disorders of the bladder, great importance has lately been attached by Dr. Ducheune (de Boulogne), who adduces them as an argument in favour of his views, that the sympathetic nerve is diseased in progressive locomotor ataxy.[1]

As to the urine itself, in all these cases, it was generally found normal, sometimes loaded with lithates, but never containing albumen or sugar.

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[1] See a paper by Dr. Duchenne: "Recherches Cliniques sur l'état pathologique du grand sympathique dans l'ataxie," etc. . . . in *Gazette Hebd. de Méd. et de Chir.*. Nos. 8 and 10. 1864. Paris.

It is remarkable that with this nearly constant implication of the bladder, the rectum should escape with impunity, or nearly so. Constipation, it is true, was complained of, as a rule; but the inactive habits of the patient sufficiently accounted for this slight sluggishness, which contrasts with the obstinate costiveness which usually accompanies ordinary paraplegia. In the case of the only female patient who has yet come under my observation, menstruation was regular, and of normal quantity. The same observation has been made by Friedreich.

One of the chief grounds on which rest the claims of progressive locomotor ataxy to be regarded as a distinct affection, apart from ordinary chronic myelitis, is decidedly the development during its course, and, in many instances, the manifestation from the very commencement, of ocular disturbances, depending either on an affection of the nerve of sight itself, or on one or more of the motor nerves of the eyeball, or on both these causes together. We accordingly find that all the patients, in the above group of cases, presented at some time or other impairment of vision or paralysis of some of the muscles of the eyeball. In case 5, however, the only abnormal condition about the eyes was that of the pupils, which were contracted to the size of pin's heads. With regard to the remaining seven cases, we find in four of them impairment of sight, varying from the mere presence of black spots in the field of vision and occasional mistiness, to actual, persistent amblyopia, and commencing amaurosis with attacks of temporary blindness. In two cases (Nos. 1 and 4) we find strabismus and ptosis; the suddenness with which these affections may set in, as well as their temporary, transitory character, being well exemplified in case 4. This patient became suddenly affected with ptosis and strabismus, which disappeared almost completely after a few months, whilst a resident in the hospital; and five years previously he had been attacked in the same way, but on the right side. In one case only (No. 7) was there simultaneously an affection of the optic nerve as well as of one of the motor nerves of the eye. This combination seems to be comparatively infrequent, for Dr. Topinard has found it in 21 only out of 102 cases of progressive locomotor ataxy, whereas vision alone was impaired in 49, and one of the motor nerves alone was affected in 51 out of the same 102 cases. With regard to the motor nerve which is most frequently affected, Dr. Duchenne (de Boulogne) has met with paralysis of the sixth cranial pair more frequently than of the third; but Dr. Topinard believes that the reverse obtains, and in the above series of cases we find the third pair affected twice against the sixth once. In cases 4 and 7, the patients when mentioning their diplopia, added, curiously enough, that one object was seen on a much higher level than the other, and not

side by side. Dr. Topinard speaks of two still more curious phenomena; namely, triplopia and monocular diplopia.[1]

The condition of the pupils was carefully and repeatedly watched in the above cases, but the facts noted are not in accordance with those published by Dr. Duchenne (de Boulogne). Thus I have never found the pupils, however contracted they might be, refuse to dilate under the influence of atropine or belladonna, nor have I been able to detect any dilatation of the contracted aperture during a paroxysm of pain. In none of these patients either were there injection of the sclerotic or conjunctiva, and a sense of increased temperature in the eye. Dr. Duchenne, however, has published cases in which these phenomena were present in the intervals between the pains, and disappeared during the paroxysms. From their resemblance to some of the results obtained by Professor Claude Bernard[2] in his experiments on the cervical portion of the sympathetic, Dr. Duchenne concludes that this nerve must be diseased in progressive locomotor ataxy; for it is well known that the division of the sympathetic nerve in the neck is followed, among other phenomena, by a contraction of the pupil on the same side as the injured nerve, and increased vascularity and temperature of the eyeball, whilst irritation of the upper extremity of the divided nerve causes dilatation of the pupil, constriction of the ocular vessels, and a diminution of temperature.

But an important question arises here: Does dilatation of the pupils, during a paroxysm of pain in progressive locomotor ataxy, indicate disease of the cervical portion of the sympathetic, or the reverse? According to Dr. Auguste Voisin,[3] if any portion of the skin of the upper extremities or of the trunk, or, better still, of the lower extremities, be pinched, pricked, galvanized, or otherwise irritated, dilatation of the pupils immediately follows. The channel of communication between the skin and the cervical portion of the sympathetic, from which arise the nerves which govern the dilatation of the pupil, would appear, from the experiments of Professor Claude Bernard, to be the anterior roots of the two upper dorsal nerves. The absence of dilatation of the pupil, therefore, when any painful impression is made on the lower extremities, may be regarded as pointing to disease either of the cervical

[1] Topinard: "De l'Ataxie locomotrice," etc., p. 160.

[2] Claude Bernard: "Leçons sur le Système nerveux," vol. ii. p. 529, and following. Consult also "Lectures on the Central Nervous System," by C. E. Brown-Séquard, M.D., p. 139 and following; and the two interesting tables there given of the effects of the section and those of the galvanization of the cervical sympathetic, with the names of the authors who first made the several observations.

[3] See *Gazette Hebdom. de Méd. et de Chir.* Paris. 1864. No. 38, p. 633.

portion of the sympathetic itself, or of the anterior roots of the two upper dorsal nerves, or of that portion of the spinal cord from which they emerge. Of three ataxic patients examined in reference to this point by Dr. Voisin, in one only the pupil, which was of medium size, refused to dilate.

The following case, published by Dr. Donnezan,[1] of the Val-de-Grâce Hospital, whilst giving anatomical proofs that the cervical sympathetic may be diseased in progressive locomotor ataxy, does not however decide the question whether the contracted pupil dilates, and the injection of the conjunctiva disappears, during the paroxysms of pain.

Mr. M——, aged 52, first complained six years ago of pain and weakness in the lower extremities. He took part in the Italian campaign notwithstanding, but his gait gradually became more and more uncertain. In September, 1860, he was admitted into the Val-de-Grâce Hospital, and on being examined, was found capable of executing simple movements with his lower limbs, when lying down, but unable to perform complex movements with precision and regularity. He complained of a sensation of circular constriction of the trunk and of tingling in the lower limbs. Sensibility to touch was diminished, but the sensibility to pain, temperature, and deep pressure was normal. There was a diminution of electro-cutaneous sensibility, but none of electro-muscular contractility. In 1863, the movements of the upper limbs were markedly ataxic. In 1864, he was on several occasions seized with sudden loss of consciousness, followed by impairment of the intellect, strabismus convergens of the right eye, contraction of both pupils, congestion of the conjunctival vessels, convulsive attacks with coma, and incoherence of language. After a time, the right side of the face became very red and ulcerated superficially, the conjunctiva swelled, and all the other symptoms growing worse, the patient died.

Dissection disclosed injection of the vessels of the spinal pia-mater and some thickening of the arachnoid, together with the usual pathological changes in the posterior columns of the cord. The cervical ganglion of the sympathetic was hard and tough, and of a peculiar yellowish-white colour, instead of being, as in health, of a pink tint, and of elastic consistency. Under the microscope, however, no difference was found between this ganglion and a healthy one. But in the ramus communicans, the nerve-substance was replaced by so abundant a lamellar tissue that it resembled tendinous tissue, and many sections had to be made before any nervous structure could be detected, and even then in an altered condition.

In one of my cases (No. 4) the left sympathetic seems to have been involved in its cervical portion; for whenever the patient was seized with pain, the left side of his forehead was covered with large drops of perspiration, whereas the corresponding portion of the face, on the right side, remained remarkably dry. The left pupil, however, which was always larger than the right (and strangely enough, for there was partial ptosis on that side), did *not* dilate any further during the paroxysm.

In the above series of cases, no other cranial nerve besides those

[1] See *Gazette Hebdom. de Med. et de Chir.* Paris. 1864. No. 19, p. 309.

of the orbital cavity seems to have been affected, unless the tremulousness of the muscles of the lower lip and chin, which T. W. (case 4) presented at one time whenever he spoke, be regarded as depending on the implication of some of the branches of the facial nerve at their periphery. Dr. Topinard, who mentions a case similar to the above, observed by himself, would probably incline to that opinion. But a much more striking instance of this complication is the one quoted by the same author, namely that of a woman who made involuntary grimaces (whenever she moved the muscles of her face, as in the act of speaking), which were exaggerated by all her efforts to control them. In none of my cases, again, was there at any time a defect of articulation, and Dr. Duchenne (de Boulogne) has even regarded the absence of this symptom as distinguishing progressive locomotor ataxy from general paralysis of the insane. But this opinion can no longer be maintained, for seventeen instances of defective articulation in ataxic patients have been collected by Dr. Topinard, who adds that in one of these cases which had come under his own observation, the patient stated that his tongue occasionally placed itself sideways in his mouth. In another of these cases (one of Friedreich's) the patient was subject to attacks of complete, although transitory, glossoplegia.

The pathognomonic symptom of progressive locomotor ataxy, namely the peculiar deficiency in the power of coordinating voluntary movements, was extremely well marked in the above eight cases. With the exception of case 7, the arms were affected in all, and the movements of the hands lacked their usual precision and regularity. This defect was manifest even when the patients kept their eyes fixed on their hands; and if they were asked to pick a small object off a smooth and level surface, their fingers could be seen to separate from one another instead of acting in concert, whilst their hand often went beyond the mark in a jerked, abrupt manner. These peculiarities became still more striking when the movements were not guided by sight. Thus, when their eyes were closed, they could not succeed, until after many repeated failures, in touching the tip of their nose with the forefinger. The arm was raised in an angular, clumsy, and abrupt manner, whilst the finger would either go and touch the forehead, the bridge of the nose, or even one of the malar bones. The loss of coordination in the arms set in gradually in all cases, and the more delicate movements performed by the hands were the first to become impossible. This defect was, in all cases, also, preceded by a sense of numbness in the fingers and palm of the hand; but until it had become highly marked, the tactile sensibility of the parts was not appreciably impaired, even when carefully tested with the æsthesiometer. This fact is of extreme importance as bearing on the

relation of loss of coordinating power to loss of tactile sensibility; and in case 6, which offered an unusual opportunity for determining this point, as the patient came under my observation within six weeks after the first manifestation of the symptoms of ataxy, no impairment of tactile sensibility was detected: the two points of the compasses were distinctly felt within normal limits, and there was no retardation in the conduction of the impressions to the sensorium. Yet the patient complained of numbness of the hands and ulnar half of the fore-arms, and the incoordination of the movements of his hands was tolerably marked. Now, however, the sensibility to touch of the skin and the general sensibility of the deeper portions of this patient's hand are considerably diminished, whilst its movements are much more irregular and incoordinate.

But it was in the movements of the lower extremities that the ataxy was most strikingly apparent, although it did not from the beginning present its peculiar and distinctive characters. All the patients spoke distinctly of a kind of premonitory stage, extending over a variable period of weeks and even months, during which *heaviness* was complained of, soon afterwards followed, whilst walking, by frequent jumpings and startings, and sudden *catchings-up* of the legs, as they often termed it, giving them the sensation of walking on springing-boards or on india-rubber balls. These sensations were doubtless owing to involuntary spasmodic contractions of separate groups of muscles, and when they affected the flexors they sometimes caused the patients to fall down. Numbness, in all the cases, preceded the incoordination; but it seemed to be a subjective sensation, which it was not possible to measure. At all events, it was distinct from real anæsthesia, for, if some of the patients declared that they could not, from the beginning, feel the ground well, many of them, on the contrary, positively maintained that they never had such difficulty, and had never known the sensation of a pad intervening between their feet and the ground. C. Y. (case 3), in whom the first indication of disease was an inability to stand with his eyes closed whilst washing his face in the morning, maintains that he had no plantar anæsthesia at the outset, although it manifested itself after the lapse of a few months. In case 6, again, the ataxic character of the walk was highly marked, and although numbness was complained of, the patient felt the ground well, and had no sensation of an intervening pad, whilst measurement with the compasses detected no diminution of tactile sensibility. After this sensation of *heaviness* had existed for some time, the patient being all the while conscious of being obliged to make unusual exertions for executing certain movements, and complaining of getting easily tired, the ataxic character of the voluntary movements became fully developed.

In some cases, as in Nos. 1 and 7, the patient discovered that he could not run, to his great surprise. In others, the disorderly and abrupt manner in which their legs were thrown about, was noticed by their friends, on whose toes they trod or whose legs they kicked when walking by the side of them. Their rolling and staggering also, their constant tendency to stumbling, excited attention, and, generally by chance, one night they found out that they were unable to walk in the dark.

The influence of *sight* in correcting to some degree, but not entirely, the incoordination of the patient's movements, was well shown in all the above cases. But another influence was also observed—namely, that of *light*. Thus, a patient who, as soon as he closed his eyes, oscillated from side to side, lost his balance and fell, managed to stand, and even walk, although he was prevented from seeing his legs by holding a sheet of paper under his chin. True, his gait was considerably more uncertain than when he could keep his eyes on his legs or on the floor immediately before them, but he yet managed to walk. Dr. Jaccoud,[1] who draws attention to this fact, and mentions Eisenmann as having been the first to note it, does not attempt to give an explanation of the phenomenon. Can it not, however, be sufficiently accounted for by the greater confidence which the patient feels when his eyes are open? The least excitement or emotion is well known to increase the incoordination of the movements of ataxic individuals, and I have often seen such patients made so *nervous* by the presence of strangers who were watching their manner of walking, that they have been unable to take more than a few steps, whereas in calmer moments they could walk all the length of the ward. The influence of confidence was well exemplified again by those same individuals being able to walk if they could take hold of another person's hand, from which they surely derived no real support.

Anæsthesia, both superficial and deep-seated, is such a prominent symptom in an advanced stage of progressive locomotor ataxy that it has by some been regarded as the essence of the disease and the cause of the incoordination. With regard to cutaneous anæsthesia, however, we find that in two of the above cases (Nos. 6 and 7) the ataxic gait of the patient was very characteristic when he first came under observation, although no appreciable impairment of tactile sensibility could be detected with the æsthesiometer. On the other hand, in six of the eight cases, the sensibility to touch was considerably diminished, this diminution being made apparent, even without measurement with the compasses, by the interval which elapsed in some

[1] Jaccoud: "Des Paraplégies et de l'Ataxie du Mouvement." Paris, 1864, p. 645.

cases between the moment when the impression was made, and that of its appreciation by the individual. In some cases, again, there was a perversion of tactile sense, as shown by the patients erroneously localizing the point touched.

But whatever be the condition of cutaneous sensibility in progressive locomotor ataxy, both physiological experiments and pathological cases completely disprove the opinion that ataxy is the consequence of cutaneous anæsthesia. Professor Claude Bernard[1] found, many years ago now, that frogs, the four limbs of which had been skinned and been therefore deprived of their cutaneous nerves, could still jump with the same precision and regularity as before; and that a hawk could continue to use its talon well, after its cutaneous nerves had been divided above and behind the metatarsal bone. In cases of plantar anæsthesia occurring in man, there have been only noted oscillations of the trunk in the standing posture, and a difficulty in maintaining the equilibrium of the body, but never that irregular and spasmodic contraction of the muscles in front of the leg, which is so marked in an ataxic individual who is made to stand for a while. Besides, Heyd (of Tubingen), who artificially produced plantar anæsthesia by enveloping the feet in compresses steeped in chloroform, found that the oscillations in such cases were infinitely less considerable than in progressive locomotor ataxy.

As to deep-seated anæsthesia, it was absent in several of the above cases, the patients positively declaring that they could feel a deep pressure well. In some of the cases, however, this condition was well marked, but yet the incoordination of the patient's movements could not be ascribed to it. The results obtained by Professor Claude Bernard,[2] after the division of the posterior roots of the spinal nerves distributed to the hind limbs of a frog, cannot be said to have settled the question at issue, because, although this eminent physiologist speaks of a want of precision and harmony in the movements performed after the operation, it appears that complete insensibility of a limb only causes a diminution in the energy of its movements, but does not impart to them the abrupt, jerking character of real ataxy. Besides, other experimenters have stated that the division of the posterior spinal roots does not interfere much with the movements of the animal experimented on, as will be seen from the following extract from Dr. Brown-Séquard's[3] lectures on the Central Nervous System.

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[1] Claude Bernard: "Leçons sur le Système Nerveux." Paris, 1858; vol. i., pp. 251, 254.

[2] Ibid.: loc. cit., vol. i., pp. 250 and following.

[3] 'Lectures on the Central Nervous System," by C. E. Brown-Séquard, M.D. Philadelphia, 1860, p. 9.

T. W. Arnold[1] has tried to show that the anterior roots of nerves contain the nerve-fibres which convey to the sensorium the impressions which give the knowledge of the state of the muscles. The chief fact on which he grounds his opinion is, that after the section of the posterior extremities of a frog it can make use of its hind legs almost as well as if nothing had been done to the posterior roots. This experiment is certainly of some value, and we must acknowledge that it is difficult to explain it otherwise than Arnold has done. Moreover, we have found that, after the section of *all* the posterior roots of the spinal nerves in frogs, the voluntary movements seem to be very nearly as perfect as if no operation had been performed, and that if the skin of the head is pinched on one side, the posterior limb on the same side tries to repel the cause of the pain, as if no injury had been made. I have also ascertained that in frogs rendered blind, these experiments give the same results. It seems very probable, from these facts, that there is at least a part of the sensations giving to the mind the idea of the state of a muscle which passes along the anterior roots to go to the sensorium.

In all the above cases, there was noted, after a time, an inability on the part of the patient to tell the position of his limbs without first looking at them. In five cases, this inability was absolute, but in cases 4 and 7, the patient could point accurately to the direction in which his limb was lying after he had contracted the muscles of that limb. This notion of position used to be ascribed to that peculiar modification of sensibility with which muscles are endowed, and which, since the days of Sir Charles Bell, has been raised to the dignity of a sixth sense, the *muscular sense*. But it is now becoming generally admitted that this notion is derived from more complex sources, and that it is made up of impressions obtained from cutaneous sensibility and the common sensibility of the deep-seated structures, bones and ligaments, as well as muscles themselves. This fact was clearly shown in cases 6 and 7, in which the sensibility to touch was not appreciably impaired, whereas in case 4, it was only slightly diminished. The patients only became aware of the position of their limbs, after they had moved them of their own accord, that is to say, after they had obtained certain impressions from the sensibility of the skin (though a certain amount of friction with their clothes, or the bed on which they were lying) and also from the stretching of the ligaments and the rubbing of the articular surfaces of the bones against one another. That ataxy is not dependent on the loss of this faculty, however, is clearly shown by the fact of the patient never losing the notion of the attitude of his upper extremities, however incoordinate their movements may be.

In six of the above cases, there was a decided and striking diminution of *electro-muscular sensibility*, that is to say, of the

[1] Ueber die verrichtung des Rueckenmarksnerven, &c. Heidelberg, 1845 Analysed in the *British and Foreign Medical Review*, April, 1845, pp. 558-575.

sensation of pain produced by the passage of an electric current through a muscle. In two alone (Nos. 6 and 7) this sensibility was apparently normal, and in them the disease had existed for a very short period only. If we admit, therefore, with Dr. Duchenne[1] (de Boulogne), that electro-muscular sensibility decreases *pari passu* with the degree of muscular sense, and that the one may be regarded as the equivalent of the other, the inference must be that there was in those cases a diminution of muscular sense. But it is more than doubtful that the sensibility of a muscle to pain is really a gauge of the degree of muscular sense present. A much better test is afforded by the faculty which we possess of appreciating differences in weight. The well-known researches of Weber have determined the normal limits of this faculty as regards the upper extremities, and they have established that it is possible, without previous practice, to distinguish the difference between two weights placed successively in the hand, when they are to one another in the proportion of 39 : 40. Quite recently, Dr. Jaccoud[2] has applied Weber's method to the lower extremities, and the results of his experiments, made on 24 different individuals, tend to show that it is possible, in health, to distinguish on an average a difference of from 50 to 70 grammes (say about 2 ounces) between two weights successively suspended from a lower limb. In six ataxic patients, to whom this test was applied, the minimum of the difference in weight appreciated by the patient was found by Dr. Jaccoud to be considerably above the health average, varying from 100 to 3,000 grammes (from $3\frac{1}{4}$ ounces to 4lb.).

Now, in two alone of the above cases (Nos. 6 and 7) was the result of the test of weight doubtful; in the other six there was decidedly an impairment of this faculty, both in the upper and lower extremities. There can be no doubt, therefore, that in the majority of cases of progressive locomotor ataxy, and probably in all, in the later stages of the disease, there is a diminution of muscular sense. It does not follow from this, however, that locomotor ataxy is merely the consequence of a loss of muscular sense. Individuals, in whom this peculiar sense is abolished, are either unable to move their limbs at all without looking at them, or, if they begin a movement, are unable to complete it, and they are unconscious of having moved. In progressive locomotor ataxy, the patient is always able to contract his muscles with his eyes shut, and when he is sitting or lying down, he is always able, if required to do so, to flex or extend his limbs, to separate or approximate them. He performs these

[1] Duchenne (de Boulogne): "Electrisation Localisée," p. 392.
[2] Jaccoud: loc. cit., p. 672.

movements in a clumsy and irregular manner, it is true, abruptly and with a jerk, but he still continues to execute them. And what is very important, he is besides conscious that his muscles are contracting, although he cannot regulate the degree of their contraction. It cannot, therefore, be said that muscular sense is abolished in such cases, unless we are prepared to admit, with Dr. Duchenne (de Boulogne), that muscles are endowed with a special faculty, distinct from muscular sense, in virtue of which they may be made to contract, independently of sight, in obedience to the will. This faculty Dr. Duchenne at one time proposed to designate by the term *conscience musculaire*, but now prefers to call *Aptitude motrice indépendante de la vue*. What is observed in progressive locomotor ataxy would seem to favour Dr. Duchenne's view, although it is more probable that the retention of the aptitude for executing a movement, without the aid of sight, merely indicates that muscular sense is not completely lost.

A capital distinction, however, between progressive locomotor ataxy and mere loss of muscular sense, is afforded by the influence of *sight* on the movements. In the latter of these affections the patient is not only able to move his limbs *when he looks at them*, but he can then perform all kinds of combined movements with the utmost precision and regularity. In the former, on the contrary, although sight will, to a certain extent, correct and moderate the disorder and irregularity of the patient's movements, it can never impart to them the harmonious co-ordination of health. However intently an ataxic individual may keep his eyes fixed on his lower limbs, he will not, when walking, prevent his rolling from side to side, nor will he escape the forcible and sudden extension of his leg in the second stage of progression, and the heavy and noisy bringing down of his heel on the floor, a symptom so highly characteristic of ataxic patients, that it has gained for them, at Gräfenberg, according to Eisenmann, the epithet of *trampers*.

The following case, which I had occasion to observe at the Westminster Hospital, where the patient was under Dr. Fincham's care, well illustrates the above remarks. There were complete analgesia, nearly complete anæsthesia, and probably, also, loss of muscular sense, although the crucial test of the appreciation of differences in weight was not applied. The movements performed were not wanting in precision and regularity, however, when they could be *guided by sight*.

W. P. ——, aged 43, married, a plumber and gas-fitter, was admitted into the Westminster Hospital, under Dr. Fincham's care, on April 25, 1865. He is tall, fairly nourished, with a sallow complexion, and there is a faint blue line along the edge of his gums. He has never had gout, rheumatic fever, or

syphilis. A year ago, he had an attack of lead colic, followed by dropping of the left wrist, which only lasted a few days.

Present state:—There is complete *analgesia* of the whole left half of the body, exactly limited to the median line—namely, of the left half of the head, face, tongue, palate, neck, trunk, and penis, and the left arm and leg. Pricking and pinching, except in the spots to be presently mentioned, are obscurely felt as a mere contact, and many seconds after the impression is made. The left eyeball is so insensible to pain that the patient rubs it with impunity. The left half of the tongue cannot distinguish sapid substances, and the patient complains of a sensation of heat and dryness, and occasionally of pins and needles in that side of the organ. Instead of analgesia, there is tenderness on pressure in front of the left elbow-joint, and again near the lower edge of the Deltoid. Differences of temperature are more acutely perceived on the affected than on the healthy side. There is nearly complete *anæsthesia* of the left half of the body, for when any point is touched on that side, the patient becomes conscious of it, only after an interval of several seconds, and besides, localizes the impression erroneously. He states that he feels as if through a thick layer of flannel or wool. He does not know the position of his left arm or leg, if he does not see it. If, when his eyes are shut, he be asked to touch the tip of his nose with his left hand, he visibly makes considerable efforts, but his hand which is raised with great difficulty and slowness, either stops at a certain height, or goes to one side of the head, never succeeding in touching the nose. The movement is slow and hesitating, never abrupt or jerked. When his eyes are shut, he cannot make the least movement with his left leg. When he walks, he keeps his eyes fixed on the ground immediately before him, not on his legs. His gait is somewhat uncertain and unsteady, but not markedly so. He cannot walk with his eyes closed; he cannot stand, even with his eyes open, when his feet are closely approximated and he looks straight before him. If, while his feet are wide apart, he shuts his eyes, he immediately oscillates from before backwards, and threatens to fall down. He says that he then feels as if he had only one leg.

There is no real *diminution of motor power* at present, at least beyond a slight depression of the left angle of the mouth, and a scarcely perceptible deviation of the apex of the tongue to the left side. There is no difference of size between the muscles of the right and left limbs; electro-muscular contractility is perfect, on both sides, but electro-muscular sensibility is almost *nil* on the left. None of the senses are affected, except *taste* in the left half of the tongue. Vision is good; the left pupil is, however, appreciably smaller than the right. Hearing is perfect on both sides. The intellect was never affected; memory is very good, and the man gives an excellent account of himself. There has never been any headache, but more or less giddiness throughout. Articulation thick and embarrassed, probably owing to the numbness of the left half of the tongue and soft palate; but the faculty of language is unimpaired. Appetite good; digestion easy; bowels at present regular, but were formerly very costive. The bladder was never affected.

Mode of Attack.—The patient's illness dates from the beginning of September, 1864. It set in suddenly, without any premonitory symptoms, about seven o'clock one morning, with a sense of chilliness and numbness all down the left half of the body. He was not, however, prevented from attending to his usual occupation, until three weeks afterwards, when the numbness was replaced by complete insensibility. There was also some motor paralysis at first, as it appears from his statement; for he affirms that there was real and considerable weakness of his left arm and leg, that his left cheek was pendulous, and that the right angle of his mouth was pulled upwards and outwards whenever he spoke or laughed. Until he was examined by Dr. Fincham, he had not discovered his inability to move his left arm and leg unless he looked at them. Under the influence of faradization, and the administration of iodide of potassium for a short time, and afterwards of hypophosphite of soda, he gradually improved,

the anæsthesia and analgesia became less and less, he had to keep less constant watch over his left limbs, and when he left the hospital, at his own request, he was considerably better.

The sensibility to *pain* is said to be often impaired in progressive locomotor ataxy, and it certainly was so in nearly all the above cases, if we judge of it merely by the sensation felt on pinching the skin; but it was not so, if we estimate it by the pain caused by pricking, for with the exception of case 8, the prick of a pin was felt as acutely in the lower limbs as in any other part of the body.

The faculty of appreciating differences of *temperature* was in no case affected, and in one instance (case 4) it seemed even to be exaggerated at one time, merely warm water giving the sensation of very hot or nearly boiling water, and on one occasion, the patient, having been ordered to take a warm bath, screamed with pain on entering it, and complained that the water was of boiling heat, although the thermometer only indicated 98° Fahrenheit.

In every one of the above cases, the amount of muscular power possessed by the patient was very considerable; and all of them, having been inclined to ascribe the incoordination of their movements to weakness, were strangely surprised to find that they could successfully resist all my efforts to bend or extend their limbs against their will. Even when the incoordination was so great that the patient (as in case 5) could not stand with his eyes open, unless he had a point of support, and could not walk except with a pair of crutches, the strength which the muscles could be made to exert was peculiarly marked. In all these cases again, the absence of all wasting and of defective nutrition, and the healthy aspect of the limbs, were remarkable.

As to the results of treatment in the above cases, they unfortunately bear out what has been found elsewhere, namely, that as yet no case of progressive locomotor ataxy has been cured. But, whilst showing that this desirable end is yet to be attained, they teach the consoling lesson that properly directed medical treatment may alleviate the patient's misery, and probably retard the progress of the disease. The influence of good food and of a highly nutritious diet was strikingly manifest in all the above cases. In fact, the treatment by tonics, which was at various times adopted, was the one which seemed to give the most favourable results. Thus, the administration of cod-liver oil in particular, with or without the syrup of the iodide of iron, the ferro-citrate of quinine, and whenever there existed a considerable degree of debility, the exhibition of ammonia and bark, were always followed by a marked improvement of the general health; and whenever such improvement could be obtained, there seemed

also to follow some diminution in the degree of incoordination. Against the pains, which sometimes constituted such a distressing symptom, belladonna was generally found beneficial, although it sometimes failed completely, after having procured the same patient relief on previous occasions. The extract of Cannabis indica, given in from a quarter to half grain doses three and four times a day, was often found of much greater value. Faradization, in some instances, chiefly in case 2, was attended with benefit, when there was marked cutaneous and muscular anæsthesia. By diminishing the degree of anæsthesia, it thereby lessened the influence of one of the causes which augmented the incoordination.

Nitrate of silver was fully tried in six of the above cases (1, 2, 3, 4, 5, and 7), but although some amelioration could be traced to it, in two cases only (1 and 4) was this appreciably marked. In the first of these cases, the patient persisted in the use of the drug for several months at a time, only leaving it off at intervals, in order to avoid the slate discoloration of the skin incidental to a saturation of the system with a salt of silver, and the result has been a marked diminution of the ataxy. This improvement has been lasting, and not merely temporary; so much so, that I have lately heard that this patient could walk without a stick. The patient, T. W. (case 4), was at one time entirely confined to his bed, and owed his marked improvement to the administration of this salt. In an interesting essay[1] by my friend, Dr. W. Edwards, of Port-Louis, Mauritius, there will be found several cases of progressive locomotor ataxy which have improved under this treatment; among others, two well-marked instances of the disease which had been under Dr. Vulpian's care.

Recommended many years ago by Professor Wunderlich, of Stuttgart, for the treatment of what he called progressive spinal paralysis, nitrate of silver acquired considerable reputation as a successful remedy in progressive locomotor ataxy, after the publication, in 1862, of a memoir[2] by Drs. Charcot and Vulpian, in which five well-marked cases were related which had considerably improved under the influence of this drug. The hopes raised by this memoir, however, have not been fulfilled. Our own cases show a very marked improvement in one case, and a less marked one in another, but no appreciable result in other instances. On the other hand, Dr. Topinard, in his excellent monograph on progressive locomotor ataxy, gives the following table of 17 cases observed by himself, in which the nitrate of silver had been tried:—

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[1] W. T. Edwards: Thèse de Paris. 1863.

[2] "Bulletin de Thérapeutique." 1862.

In 12 cases, the drug failed completely.
„ 1 „ there was a relative cure.
„ 1 „ there was a very relative, but rapid improvement.
„ 1 „ the improvement was pretty well marked, but of short duration.
„ 1 „ it was slight and very transitory.
„ 1 „ it was doubtful.

The inference which Dr. Topinard draws from this, and the conclusion which he therefore adopts is, that "nitrate of silver is, in general, of no efficacy in the treatment of progressive locomotor ataxy.[1]"

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[1] Topinard : "De l'Ataxie Locomotrice," &c., p. 459.

LECTURE VII.

ON APHASIA.[1]

There is not only loss of speech, but loss of memory also:—1. Amnesia of speech; 2. Amnesia of speech and writing; 3. Amnesia of speech, writing, and gesture; hence, three principal forms of Aphasia.—Transitory Aphasia and persistent Aphasia. Special Anatomical lesions of Aphasia. Function of the posterior portion of the third left frontal convolution. Unique case in which the seat of the lesion was probably on the right side.—More or less profound, and undeniable disturbance of the intellect, in Aphasia.

Gentlemen,—Some of you may recollect a young mechanic, about twenty-five years old, who occupied bed No. 2 in St. Agnes Ward. He had walked to the hospital, he was not lame, he used both his hands perfectly, his face was full of intelligence, and yet he was not able to answer any of my questions, although his tongue was very mobile. He heard me well and looked at me whilst I questioned him; his gestures, his looks, showed that he understood all I said; it seemed as if his mind were full of thoughts which he could not express in words. He knew how to read and write, and yet when I gave him a pencil and some paper, and asked him to write his name down, he held the pencil properly but only wrote meaningless letters, and then threw away the pencil in a fit of impatience. He, however, remembered a few words, which he kept constantly repeating, showing at the same time by his manner, that he well knew how little those words expressed his meaning. His illness had set in suddenly, after certain excesses.

It was evident that some local changes had occurred in the structure of his brain, which were certainly not due to hæmorrhage or softening; and as there was no headache and no fever, I waited until more light could be thrown on the case. But before a fortnight had elapsed the young man recovered completely, without having been subjected to any treatment, and was able to leave the

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[1] The affection which I am about to describe was, in 1841, termed *alalia* by Professor Lordat; and in 1861, Mr. Broca changed this name for that of *aphemia*. But Mr. Chrysaphis, a very distinguished Greek scholar, and a Greek himself, although accepting the term *alalia*, proposed, however, as a better one that of *aphasia*, derived from *a* privative and φασις, speech. Mr. Littré, whose authority is so great, and Dr. Briau have likewise preferred the word *aphasia*, and all three concur in rejecting *aphemia*. I had at first adopted the name of *aphemia*, after Mr. Broca, but I have now, on the authority of the savants whom I have mentioned, substituted for it that of *aphasia*.

hospital. Every day a new word was added to his vocabulary; then incomplete or incoherent sentences were succeeded by very connected ones, and at last, he managed to hold a conversation, although he from time to time felt some hesitation, and was sometimes unable to find the exact word which he wanted to express his thought. By the time that he left the hospital, all abnormal symptoms had disappeared. He had previously been able to give a pretty good account of what had passed in his mind. He well knew that he had lost all recollection of words, and he was besides conscious that his mind was not then so clear as it had been.

On the 27th of February, 1861, the woman Desteben, aged fifty-eight, was admitted into the St. Bernard ward. After she had left the hospital, she was admitted on the 12th of April following into the Salpêtrière, where she died on the 16th of April, 1863, of what is called *cerebral congestion* in the register, but no post-mortem examination was made. During the forty-four days which she spent at the Hôtel-Dieu, this woman was most carefully watched, and I remained every day a pretty long time by her side. I learnt that on several occasions, after slight apoplectiform seizures, she had experienced considerable difficulty in speaking, although she was not in any way paralysed. When I saw her, the movements of her limbs were perfectly free; she moved her tongue about with as much facility as any other person, but she could not articulate anything besides, "Oh! how annoying!" It was thus she expressed her impatience on finding her inability to answer my questions. Although she looked intelligent, and behaved in the ward like a sensible person, I could never obtain another word from her. She knew perfectly the use of the objects which I pointed out to her, but she was never able to tell their name; and if an attempt was made to deceive her, by calling the object by a wrong name, she protested very clearly by her gestures, whilst she on the contrary made signs of approval, when the true name was mentioned. As she did not know how to write, it was impossible to ascertain the manifestations of the intellect which might have been revealed in writing.

This patient was remarkably clean in her habits: she made her own bed, combed her hair, and dressed herself with a certain amount of vanity, put away carefully all her toilet articles, and never exhibited, during her residence in the hospital, the least hesitation in her movements, which always remained perfectly regular.

The next case which I shall relate to you is all the more precious, from the patient being one of my most eminent colleagues in the Faculty of Medicine, and from his having paid special attention to the study of cerebral diseases. Mr. X—— had been

confined to his bed for a few days in the country in consequence of an injury to one of his legs; and being alone there, he read all day, and thus fatigued his brain. He was engaged one day reading one of Lamartine's literary conversations, when he suddenly noticed that he did not clearly understand what he was reading. He paused for a short time, but on beginning again, he made the same remark. Alarmed by this, he tried to call to his help, but to his extreme surprise he was unable to utter a word. Believing himself to be struck with apoplexy, he then executed various complex movements with both his hands and with his uninjured leg, and thus found that he was not in the least paralysed. He then rang the bell, and when a servant came into the room, Mr. X—— could not speak a single word; and yet he could move his tongue in every possible direction, and was perfectly aware of this singular discrepancy between the facility with which his vocal organs were moved and his inability to express his thoughts in words. He made signs that he wished to write, but on pen and ink being brought to him, he was as incapable of expressing his thoughts in writing as in speaking. As he had always paid particular attention to diseases of the brain, he tried, however, to analyse his symptoms, and to explain them by some special lesion of the brain, reasoning mentally on his own illness, as he would have done in a clinical conference. His servant immediately telegraphed to Mrs. X——, who was in Paris, and sent for a medical man, who came two or three hours afterwards. Mr. X—— then pulled up his shirt sleeve, and pointing to the bend of his arm, clearly intimated that he wished to be bled. No sooner was the bleeding over, than he was able to say a few words, although incoherently and imperfectly. Still some of these words clearly expressed an idea; whilst others seemed to have no direct relation to the principal idea. By degrees the veil was removed; he had a greater command of words to express his ideas which also became more numerous, and at the end of twelve hours recovery was complete. Let me here observe, and I shall revert to it by and by, that Mr. X—— had been suffering from diabetes for a few years.

The next case, which very much resembles the preceding, is that of a gentleman suffering from Bright's disease. He was sixty years old, and was sent to me on June 18, 1863, by Dr. Denoette (of Hâvre). He was a terrible sufferer from gout, and notwithstanding his doctor's advice to the contrary, had drunk to excess of Vichy water. He had therefore had internal gout, and had become extremely cachectic; for some years, he had passed albuminous urine. In 1861, he had a nervous attack, the details of which he related to me, and which were confirmed by his wife. He was playing whist at his club, and in the middle of a game, threw his cards on the table. On his then attempting to speak, he was unable to utter a single word, and yet until that

moment he had noticed nothing strange, and had played with his usual skill. Feeling alarmed, however, he gets up, takes his hat, his walking-stick, and walks home quickly, more quickly even than usual. On arriving home, he tried to tell his wife what had occurred. By that time he could say a few words, parts of sentences even, but omitting words which he could not find, and getting impatient in consequence. The difficulty which he had in expressing his thoughts increased every moment, however, and before two hours had elapsed, he was incapable of articulating a single word. Yet his limbs and his tongue could be moved as freely as in health.

The usual medical attendant of the family, who arrived by this time, prescribed leeches round the anus, and whilst the servant was gone for them, the patient's wife wished to find out whether he could read. But he could not read the newspaper which she held before him, although his sight was perfect. On my questioning him on this point, he said that he could see the letters and the words, but did not understand their meaning well. The leeches were next applied, but he seemed to grow very impatient because they took slowly and with difficulty. He tried to explain why he was impatient, but his gestures, although normal, were not understood. Speech was completely gone. Three of the leeches, however, had scarcely begun to swell, when speech returned a little; the patient could then make himself understood, but still left out a few words in each sentence. He asked for better leeches, and when fresh ones were applied and drew blood in abundance, all the strange symptoms vanished, and the patient expressed his thoughts with as much facility as ever. He then related all the circumstances of his seizure, and explained that he was so impatient when the first leeches were applied, because he wished for better ones, and was annoyed at finding that his gestures were not understood. Since that time he has had no second attack of the kind, and the Bright's disease from which he suffers, has continued without presenting any abnormal symptoms.

The two following cases have been communicated to me by my friend Dr. Voyet, of Chartres.

M. X——, a veterinary surgeon of X. (Eure et Loire) aged forty-six, of robust constitution, single; no previous illness with the exception of a cancer of the under lip, successfully removed by operation in 1863. About the end of September, acute articular rheumatism. On the 29th of October, 1863, intense dyspnæa in the middle of the night; I was sent for, and saw the patient for the first time on the morning of the 29th of October. During the paroxysm of dyspnæa, the rheumatic pains disappeared, the pulse was intermittent, and so irregular that it could not be counted; there was the same disturbance in the heart's action, but no abnormal bruit; the patient's anxiety was such

that he could not remain in bed. Sinapisms and a blister applied over the præcordial region gave immediate relief; upon which the rheumatic pains returned and the heart's action became regular again. On November the 4th, M. X—— was sitting by the fire and talking to a friend, when he suddenly stared at his friend with a stupid look without being able to utter a single word. About five minutes afterwards he began to sputter out the word *monomentif*, which he kept repeating for four hours. Annoyed at not being able to make himself understood, he made signs that he wanted pen and ink, but only traced on the paper shapeless signs, like a child who does not know how to write. Four hours afterwards, on his renewing the attempt as he wished to inform his brother of his condition, he only succeeded in writing legibly, "My dear," the rest was as shapeless as on the former occasion. By this time, however, he began to articulate a few monosyllables, but always ended them by *tif;* and if he wished to say a word of several syllables, he only pronounced the first syllable and added *tif* to it, saying, for example, montif for monsieur, bontif for bonjour, etc.

On the following day, he could answer questions that were put to him, but could not construct a sentence: yet he could be understood. On the second day, all abnormal symptoms had disappeared, whilst during the forty-eight hours that the Aphasia had lasted, there had been no trace of paralysis.

On the 12th of February, 1864, that is, more than three months after the above attack, M. X——, who still complained of rheumatic pain in the right wrist, was eating his soup, holding his spoon in his left hand, when he suddenly dropped it, and his arm became completely powerless. Dr. Voyet, who was immediately sent for, found, on his arrival, that there was paralysis of the whole left side of the body, together with great embarrassment of articulation and deglutition. By the next morning, all the symptoms had disappeared. On this occasion, the difficulty in speaking was not in the least like what had been felt in November; at that time, M. X—— could not find words to express his thoughts, whereas now he found words, but had a difficulty in articulating them.

"On February the 12th, 1864, Dr. Voyet saw in consultation Mrs. X——, aged 58, residing at Voves, and for many years afflicted with hypertrophy of the heart. He was told by her daughter, that in the beginning of February she was awakened by an unusual noise proceeding from her mother's room, and on her going there, she found her mother in the act of gesticulating and repeating constantly, '*Vousi, vousi.*' After two hours, this condition disappeared, and Mrs. X—— then related that she wanted to ask for ether, and to say that a doctor should be sent for, because she felt that something extraordinary was passing within her."

Another curious case is the following:

Mrs. B——, the mother-in-law of a medical man, had never been paralyzed, but laboured under a very singular intellectual disorder. Whenever a visitor entered her apartment, she rose with an amiable look, and pointing to a chair, exclaimed: "Pig, animal, stupid fool." ("Mrs. B. asks you to take a chair," her son-in-law would then put in, giving this interpretation to her strange expressions.) In other respects, Mrs. B.'s acts were rational, and her case differed from ordinary aphasia in that she did not seem to grow impatient at what she said, or to understand the meaning of the insulting expressions of which she made use. In the seven cases, which I have just related, the nervous phenomena seem to affect the intellect alone, and especially the aptitude for expressing one's thoughts in writing and speaking. I now pass on to more complex cases, in which there evidently is a deeper lesion of the brain, characterized by some impairment of the motor power, as well as by the peculiar phenomena to which I have just called your attention.

M. X——, a very eminent jurist, consulted me in the beginning of 1863, and his wife, a remarkably intelligent person, told me of details which the patient perhaps would not or could not have communicated to me. From time to time, in the middle of a conversation, he could not find the word he wanted, or substituted a strange one for it. On other occasions, he would call out to his wife: "Give me my—my—dear me! my—you know;" and he would point to his head. "Your hat?"—"Yes, my hat." Sometimes, again, he would ring the bell before going out, and say to the servant: "Give me my um, umbrel, umbrel, oh dear!"—"Your umbrella?"—"Oh yes, my umbrella." And yet at that very time his conversation was as sensible as ever; he wrote on, read of, or discussed most difficult points of law. He complained, however, of some heaviness of the head, and pretty frequently also of some numbness of the limbs, more marked on the right than on the left side.

Now, gentlemen, let this singular forgetfulness of words be considerably more exaggerated, and you will have Aphasia with its usual characters. In the first cases which I related to you, there existed no symptom of paralysis, but in the last one, there is very probably, if not certainly, an organic lesion of the brain. The following case, which was in my own wards, is all the more interesting from the patient being now completely cured, and from her being able to analyze herself all the phenomena which occurred in her case.

On the 1st of April, 1862, Marie K——, aged 50, was admitted into the St. Bernard ward. A month previously, she had begun to complain of certain symptoms which she afterwards described perfectly. She had violent headaches, followed, from time to time, by convulsive movements of the right half of the face

which lasted a very short time only, and left behind them a momentary embarrassment of speech. She had never lost consciousness, and she even added that she had got up during an attack, and had fetched a handkerchief in order to wipe off the frothy saliva which ran out of her lips. Two days before her admission she had had a more violent seizure, during which she had bitten her tongue.

During the first week after her admission, she could only say a few words devoid of any precise meaning. She looked intelligent, and yet she could neither tell nor write the name of the most common-place objects, such as a watch, a key, a spoon, or a fork. She could articulate her name well, and write it easily; but if, after having written her name, she was asked to write the word *spoon*, she went on writing her name, and yet noticed her mistake, as could be seen by her look of annoyance. When I insisted on her repeating a word after me, she made signs that there was an impediment in the right side of her throat, and strangely enough, another patient, Adèle A——, of whom I shall presently speak, complained of a painful tightness in the same spot.

Marie K—— read a good deal during the day, and we were all taken in by this semblance of intelligence, but after her recovery, she told us that she only read with her eyes, not with *her stomach*, a singular expression which she used, meaning thereby that she did not understand what she read. After she had been a few days in the hospital, she got better, and recovered the faculty of speech. She then told us that, in the preceding year, she had been subjected by her medical attendant to a very severe mercurial treatment. I therefore suspected that her peculiar affection was due to some grave syphilitic lesion of the left cerebral hemisphere, or of the base of the brain; and on my prescribing powerful anti-syphilitic remedies, I had the satisfaction of seeing the abnormal symptoms disappear, and her health get re-established. She has now left the hospital for two years, but she has often come here to show herself. She has for several months taken, and still takes, from time to time, iodide of potassium.

On the 9th of December, 1854, a young labourer, aged 28, was admitted into St. Agnes ward. Two days previously, according to the statement of his friends, he had been seized, suddenly and without any assignable cause, with complete mutism. His previous health had been good on the whole, he had led a regular life, and yet he had two years previously suffered from violent headache, and even been delirious. He was bled from the arm, and got well again; and since then, he had not been taken ill in the same way.

The affection, for which he had come to the hospital, consisted

solely in an utter inability to speak, although his intelligence seemed to be unimpaired, and he could perfectly understand all the questions which were put to him. But to these questions he invariably answered "*No*," even when he nodded his head to signify assent. One of the students, however, informed me that when left alone with him, he had succeeded in making him say the word "*cloak*," after many repeated trials. I found only a marked deviation of the apex of the tongue to the right, but no other sign of paralysis; the face, the trunk, and limbs, could be moved with perfect freedom and force.

The second day after the patient's admission, I ordered him to be bled, and after this, he seemed to be able to move his tongue more freely than before, but there was the same complete Aphasia. When I asked him to write his name down, he did so correctly, but when I told him to write down what had happened to him, he only wrote "*was, was, was.*" He knew perfectly well that this was not what he wanted to write, and, annoyed at not being able to express his thoughts, he put down the pen. Two days after this, on my asking him to write down the name of his birth-place, he wrote "*alone, alone, alone*," and did so again when I desired him to write "*good morning.*" His impatient gestures, all the while, showed that he was perfectly conscious that he was not writing what he had in his mind. On the following day, he wrote again words that had no sense, such as "*game*" for "*soup*," but he could say, "*Good morning, sir*," speaking, it is true, like a child who is learning to speak. A few days later, he said very distinctly: "*I am pretty well*," and then "*Good morning, sir, I am getting on well*," with a hesitating voice, however, like an habitual stammerer who endeavours not to stutter. When the attempt was renewed to make him write, he only scribbled on the paper series of syllables without any meaning, but he managed to write under dictation: "*I have eaten.*" He left the Hôtel-Dieu on the 24th of December, although there had been no appreciable change in his symptoms. He asked himself for his discharge, however, saying very distinctly: "*I wish to go away.*"

In the first cases which I brought under your notice, gentlemen, you saw Aphasia set in without paralysis. I next related to you cases in which this affection occurred together with a very slightly marked and transitory paralysis, and persisted, as in the instance of Marie K——, even after all trace of weakness of the arm and leg had disappeared. I will now pass on to cases in which the Aphasia was extremely marked, and the paralysis of longer continuance, although still transitory.

I was consulted in the year 1863 by a gentleman, a recorder, aged 49. In the month of January of the previous year, he had got up one morning in his usual state of health, and had

afterwards sat for five hours in his office, engaged in his usual occupation and without feeling any abnormal sensation. On getting up, however, he found that his right leg felt a little numb. He walked upstairs to his bedroom by himself, but on his way there, felt that his right arm was becoming affected. He spoke with facility, and dictated to his wife a very sensible letter addressed to the gentleman at the head of his department, as he was afraid that this attack of paralysis would keep him from his duties for a time. A few hours later, although the paralysis had not increased in degree, he could no longer speak. Yet he seemed to recognize and understand everything, but could only say: "*Nasi bousi, nasi bousi,*" repeating these unmeaning words whether he asked or answered a question, or pointed to anything. Eight days afterwards, he recovered completely the faculty of speech; and at the end of a month, the paralysis disappeared. This patient was obese, and troubled with piles; his heart was normal. A few days before his attack, he had felt pain in the back of his neck and the left side of his head, but he was pretty frequently liable to this pain, whenever he did any hard work. Two years previously, he had suddenly felt a kind of electric shock in his left hand, which had since then felt slightly numb. When he came to consult me, accompanied by his wife, who told me the above details, he was in the full enjoyment of his intellectual faculties, walked well, but still wrote with great difficulty. As soon as he took hold of a pen, his arm moved violently away from the trunk, and he could only manage to write, and even then with difficulty, by strapping his arm down. He was, therefore, affected with what has been called *writer's cramp,* or what Dr. Duchenne has designated by the more appropriate name of *functional spasm.* This patient was also slightly deaf on the right side, and complained of a sensation of burning in the skin of the right half of the body.

In some cases, the disease is more lasting, probably from its being due to a deeper and more persistent lesion of the brain. Thus, at No. 8 in St. Agnes ward, you can now see a patient of the name of Marcou, who is affected with Aphasia and *left hemiplegia.* Mark that I say *left hemiplegia,* and this is an important case, because, as far as I am aware, it is the only one as yet recorded, in which the paralysis has not been on the right side. This man is thirty years old. He came to the hospital on foot, but could give no information about himself, nor tell his name, occupation, and address, in the office where the names of patients are entered on a register before they are sent to a ward. His stock of words was restricted to these two: "*My faith!*" and when he was pressed hard, he looked impatient, and uttered the oath, "Cré nom d'un cœur!" A stratagem was thought of in order to find out his name and address. He was told that he could not

be admitted into the hospital, and had better go home. The poor fellow understood, and went away. He was followed, and was seen to go to a stone-yard, where he sat down on a stone. The workmen there knew him, said what his name was, and added that he had come to the yard that morning, dragging the left leg a little, and unable to speak. He was then brought back, and admitted into the hospital.

The next morning I easily recognized that he was suffering from Aphasia, when I questioned him. I asked him what his name was, and his occupation; he looked at me, and answered: "My faith!" . . . . I insisted, but in spite of his efforts, he only shook his head with an impatient gesture, exclaiming: "Cré nom d'un cœur." As I wished to find out how many words he had at command, I said to him: "Are you from the Haute-Loire?" He repeated like an echo, "Haute-Loire?" "What's your name?" —"Haute-Loire." "Your profession?"—"Haute-Loire." "But your name is Marcou?"—"Yes, sir." "You are sure it is Marcou?"—"Yes." "What department do you come from?"—"Marcou." "No; that's your name." But with an impatient gesture, he exclaimed, "Cré nom d'un cœur." His mouth evidently deviates a little to the right, owing to the paralysis of the left side of his face. When he is pressed to say what he complains of, he lifts up both his arms at the same time, but whilst he moves his right arm briskly and closes his right fist with force, he looks sadly at his left arm, which is relatively powerless, although he can still use it pretty well. When he is in bed, or even when he walks, a certain degree of attention is required in order to recognize that he is paralysed on the left side. I dare not say that he looks as intelligent as he may have done formerly, but he has not the dulness of aspect which sadly strikes us in persons who have had cerebral hæmorrhage. It is very remarkable, also, that aphasic individuals (even when completely hemiplegic) do not shed tears like those who have had apoplexy.

I cannot say what influence has been at work to bring on this man's complaint; but on examining his organs, I thought I could recognize traces of an indurated chancre cured long ago. I accordingly treated him by mercury and iodide of potassium; and after alternating periods of very marked improvement and momentary aggravation of the disease, there is at last a permanent improvement, which, however, is not a complete recovery. The poor fellow, in spite of three months' lessons and efforts, can never remember the word *hair*, and can only say *cotton* when he wants his cotton cap, although he says the word *cotton* with evident complacency.

At No. 20, in the same ward, is a man forty years of age. He has been pretty well educated, since he was at one time

destined for the clerical profession, and was in a seminary, so that it will be easier in his case to study the impairment of the intellectual faculties, and to appreciate its various manifestations. He is a married man, and a father, but he is far from having led a regular life. He is particularly addicted to drunkenness. Four months ago, after having complained of headache, which could be reasonably attributed to his intemperate habits, he had a fall in his bed-room; but his wife, who was accustomed to see him fall down after too copious libations, took no notice of it. That night she went, as usual, to bed with one of her children, but was awakened from her sleep by the noise of her husband falling down a second time, through his having got his foot entangled under a wardrobe. He got up without saying a word, his wife asking him no questions, went to the bed in which he usually slept by himself, and all through the night was violently agitated, as he used after all to be, whenever he came home drunk. In the early morning, however (it was summer time), his wife, on going to his bed, discovered that the bottom of the bed was broken, whilst he was lying almost completely naked, and messed all over. His face had not its usual expression, and when his wife rebuked him, he looked at her in a strange manner, repeating, "*Cou si si, cousisi.*" These are the only words which he has spoken for the last four months, and he keeps saying them at every turn, when he is in a passion or when he wishes to express his gratitude, when he asks for or refuses anything. When he is very excited, however, he calls out, "Sacon, Sacon," probably an abbreviation of the oath, "Sacré nom de Dieu." When the poor wife recognized the gravity of her husband's condition, she tried to help him, and then discovered that he was paralysed on the right side. Upon this, she brought him to the hospital.

You have seen him to-day, and you have found that he is less paralysed than he was, for he easily moves his right arm and leg, although he cannot at all perform those movements of the hand which require a certain degree of precision, as the act of writing for example. He can write with his left hand, however, and we shall thus be able to appreciate the state of his intellect. When I ask him his name, he answers, "*Cousisi;*" but when I ask him to write it, he writes down, "*Paquet.*" If then I wish him to write his address, he again writes, "*Paquet.*" Yet he perceives that he has made a mistake, and turns his head impatiently away, saying, "*Cousisi.*" He can write the word "*note*" when a printed copy of the word is set before him; but if this be then removed, and he be asked to write his name, he writes down "*note.*" As he had nodded assent when he was asked whether he could play backgammon and dominoes, several patients in the same ward were requested to play with him in turn, and they all declared that he played well, that he knew all the tricks of the

game, and that he even cheated when he found that he was losing. Good luck made him laugh, whilst bad luck rendered him fidgety; in either case his gestures were very significant, or he kept repeating "Cousisi."

His wife, who had come to give me any information which I might desire, had brought with her her boy who had disease of the knee-joint. Whilst I was examining the joint, the boy's father made sign to him by repeatedly touching his own forehead with his hand to remove his cap, which he had kept on his head, and the man looked vexed at this want of deference on the part of his son. I lay great stress on all these details, because they will be useful when we come to examine how far the intellect is preserved in such cases. There is another detail, however, which I must not omit. This patient has several newspapers, containing tales, which he reads, and expresses by signs that he understands them perfectly; but his neighbour states that he reads them over again every day, and several times even on the same day. He surely could find no interest in them if he understood them well, or did not forget them. I have already told you, and I attach importance to the fact, that the hemiplegia has diminished, although, according to his wife's account, the manifestations of the intellect have not become developed in the same degree.

I now pass on to a series of cases in which there has been complete and persistent hemiplegia, whilst the Aphasia has remained unchanged since the invasion of the disease.

The following case is sufficiently important to warrant my entering into minute details. I was asked to go and see the patient in the department of Landes, in the spring of 1863. His symptoms had been noted with considerable care by his relative, Dr. G. Hameau, of Arcachon, and I saw him in consultation with Drs. Sourouille, de Loustalot, Hameau, and Laffitte.

M. X—— is fifty-seven years old; he is possessed of great wealth, and has lived freely, without, however, committing any excesses. His father died of some chronic chest affection; his mother is still living, and eighty-seven years old. His grandfather, on his mother's side, and his great grandmother, died of apoplexy, the first when seventy, and the second when sixty-five years old. An uncle, on his father's side, died of apoplexy at the age of sixty-five, and an aunt, on his mother's side, died from the same cause, aged fifty-eight; whilst another aunt died recently at the age of eighty-four, in an epileptiform seizure following upon cerebral hæmorrhage.

From his early youth M. X—— has been subject to paroxysmal headaches, of great violence and long continuance, recurring two or three times every month.

When he reached the age of forty-five, his headache was replaced by fits of normal gout, for which he went to Vichy, and

drank the waters there, but without deriving any benefit from them. The attacks of gout had not been in the least modified, when three or four years after the thermal season at Vichy, M. X—— had one night an attack of vertigo, as he was going to take tea. He was standing at the time, when he suddenly felt giddy and his sight grew misty; he leant against the mantelpiece in order not to lose his equilibrium. This occurrence excited little attention, although there remained after it some unsteadiness of the right hand and a marked difficulty in writing. A few years previously, when M. X—— was only subject to headache, he once had suddenly noticed that his sight had become dim, and this weakness of sight had lasted for a month or two. It is very probable that on that occasion, as on the succeeding one, trifling hæmorrhage had occurred in the brain. Two or three years elapsed without any fresh seizure; the gouty pains returned during the winter, whilst the hand recovered very slowly its former power.

A second attack of vertigo then occurred; the patient was at the time sitting on a chair and dressing. The giddiness lasted somewhat longer than on the first occasion, and from that day the right arm became more markedly weak and the tongue slightly embarrassed. This impediment attracted notice whenever M. X—— spoke with animation.

The intellect had failed a little. During the summer of 1857, he went to Ems, and was slightly better for a few months. In 1858, he went to Bagnères-de-Bigorre; in 1858, to Dax; but in December, 1859, his condition became less satisfactory; he had some fever and pain in the epigastrium. On the 2nd of February, 1860, at 7 o'clock in the evening, he had a severe attack. As he got up to shake hands with the curate of the place, he suddenly staggered, stammered, and dropped into the arms of his visitor, who had rushed forward to support him. He remained in the most profound apoplectic stupor for more than ten hours, with complete paralysis of the right side. For a few days, he gave only very obscure signs of intelligence; but from that seizure, he entirely lost the faculty of speech. A few months afterwards, he almost completely recovered the power of moving his right leg, but the movements of his right arm have always been impeded. During the summer of 1860, he had, for the first time, an epileptiform seizure; he had three of these during that year, six in the following year, and four in 1862, up to the month of August; since which date, he has had no return of them.

When in the spring of 1863, I saw M. X——, I found him looking very well and dressed with care, and even with elegance. His face was intelligent and smiling, and full of benevolence. He rose when I entered, and showed by his gestures, and espe-

cially by the expression of his face, that he was pleased to see me. He could not speak, and only uttered in a faltering voice unintelligible words, in which the monosyllable, "*Yes*," returned frequently. When I questioned him, he answered "Yes" to everything, even when he shook his head in denial. "How old are you?"—"*Yes!*" "How far back do you date your illness?"—"*Yes!*" etc., etc. It could be easily seen, however, that he was not satisfied when the word, "Yes," was wrongly applied, for he then made an impatient gesture. He looked pleased, on the contrary, when the word was used appropriately. He sat to table with us at dinner, used his left hand, and ate with great propriety, and with infinitely greater reserve than the generality of ordinary paralytics, who often eat voraciously and in a dirty manner. He looked after his guests during dinner, and took part in some of the discussions carried on. When the delicate flavour of the lamb of the country was praised, he nodded assent; whilst, on some of the guests saying that the kid of the country had a better flavour than the lamb, he shook his head in disapproval. He made signs to the servant to hand the wine round, and when wine of an esteemed vintage was going round, he made signs that it should be drunk in preference to the rest.

The dinner over, he rose with the rest of the company, resting on his stick, and politely let the gentlemen who were escorting ladies pass first. We next sat down, and I tried to make out how far he could give proofs of intelligence. As he always answered "*Yes*," I asked him whether he knew how that word was spelt, and on his nodding assent, I took up a large quarto volume with the following title on its back, "History of the Two Americas," and requested him to point out the letters in those words which formed the word yes. Although the letters were more than one-third of an inch in size, he could not succeed in doing as I wished. By telling him to seek for each letter in turn, and by calling out its name, he managed, after some hesitation, to point out the two first, and was very long in finding the third. I then asked him to point out the same letters again, without my calling them out first, but after looking at the book attentively for some time, he threw it away, with a look of annoyance, which showed that he felt his inability to do as I wished him.

He played every day at All-fours, hiding his cards behind a pile of books, and using his left hand. He often won when playing with the curate, the doctor, or his son, without their allowing him to do so out of kindness. Whenever he played a trump, he laid his hand on it with an air of authority which showed that he knew its value. His son and Dr. Laffitte declared to me that he played as well as he ever used to do. Sometimes his son sits by his side to advise him, and stops him when he

takes a card which is not the proper one, but he insists on playing as he likes, and by winning the game proves to his adviser that if he sacrificed a card, it was because he could thus improve his game. Although his son manages all his affairs, he insists on being consulted about the leases and contracts, etc.; and the son stated to me that his father indicates perfectly well, by gestures which are understood by those habitually round him, when certain portions of the deeds do not please him, and that he is not satisfied until alterations are made, which are, as a rule, useful and reasonable.

Although his sight was good, he could not read, or, at least, understand the sense of what he read; he listened with pleasure, however, when he was read to. When I asked him his age, he told it me in such a remarkable manner that I must mention it. After first closing his left hand, he opened it out, showing me his five fingers one after the other; he then closed the hand a second time, and next stretched all his fingers out simultaneously and separating them at the same time. For a third time he closed his hand again, and then showed me two fingers. I confess I did not understand what he meant, and I said to him that he had only indicated twelve years. He laughed at this, as if he knew that the want of intelligence was on my side. He then began anew slowly to show me his five fingers, one after the other, making each time a movement with his head and arm as if he wanted to fix my attention more. On my saying that he meant to say fifty, he nodded approvingly with a smile, and then opened out his whole hand, and after closing it, showed me two fingers. He meant to say fifty-seven years; in fact, his real age.

He could not put together loose letters of the alphabet, nor write with his left hand, two things which most paralytics can do. It has often happened to him to say a word which he has not uttered for a very long time, as if an old impression were revived in his brain. Some time ago, he dropped his handkerchief, and as a lady near him picked it up and gave it to him, he said to her, "*Thanks!*" in a loud and distinct voice. His friends were delighted at this, and thought that he had recovered his speech. He was asked, implored to say the word again, it was repeated to him several times, but all was in vain, he never could succeed. I will presently speak of an English banker whom I saw with Drs. Campbell and Blondeau, and who presented anomalies of the same kind.

I now come to a most important point. It seemed as if this patient was in full possession of his intellect, and the expression of his face clearly showed this, as well as his aptitude for card playing. Yet he had forgotten the words themselves, besides having become incapable of articulating them. When I took up his spectacles and asked him what they were, he seemed to make

an effort, and yet answered only *yes* as usual. It was evident, however, that he was not satisfied with his answer; and, taking the spectacles with his left hand, he placed them over his nose, as if to show that if he could not tell the name of the object, he knew perfectly the use of it. I then asked him if he remembered the name of the article, but he shook his head. "It's a pen," I said, but he laughed, and moved his head and arm as if he understood that I was joking. "It's a knife, then," and he continued to laugh. "Perhaps it's a pair of spectacles?" *Yes*, he replied quickly, clearly showing by signs that I was not joking then. On my attempting, however, to make him say the word *spectacles*, he failed even to articulate the first syllable.

Whenever he grew impatient, he did not always show it exactly in the same way. With strangers, he contented himself with a very significant movement of the shoulder, shrugging it with a look of discouragement and *ennui*. But when he was alone with his wife, his son, or his servants, he showed his impatience by using a very well-known oath.

He sometimes proposed guesses to those about him. He seemed to take great pleasure in looking over collections of portraits in illustrated papers; and he would sometimes hide the name underneath the print, and ask whose portrait it was. As this somewhat childish game seemed to amuse him, his friends kept it up by pretending to make mistakes. "It's Napoleon's portrait," they say, "Alexander's, of Russia," or "King Leopold's of Belgium." He laughs incredulously, and continues to ask. "It's Queen Victoria's," and he then takes his hand away, showing that the person has guessed right.

The young woman who was at No. 5 in St. Bernard ward, was in exactly the same state, and the history of her case is as follows:—

Adèle Anselin, aged thirty-two, of apparently good health, stout, and with a fresh complexion, was admitted under my care whilst suffering from acute pneumonia, as well as from right hemiplegia, and on examining her with care, I recognized the presence of chronic endo-pericarditis and mitral regurgitation. In the course of the year which she spent in my ward, she had slight attacks of hœmoptysis, perhaps depending on the state of her heart; but when she left the Hôtel Dieu, her general health was as good as possible. Before she came under my care, she had been an in-patient at the Lariboisière Hospital for several months, on account of her paralysis, which had occurred two years previous to my seeing her. She had formerly had acute articular rheumatism, so that I conjectured that her cerebral affection was the consequence of an embolon, which had come from one of the diseased cardiac valves. The hemiplegia had been sudden and complete; she had not lost consciousness; but

the faculty of speech was abolished then as it was on admission. The paralysis of the arm had not been in the least modified, but she could perform certain movements with her right leg.

You remember how long I used to stand by her bed, in order to ascertain the state of her mind. When I asked her her name she could not tell it me. Whenever she got impatient, she exclaimed, "Oh, pity!" She answered pretty well by signs. When asked whether she remembered her name, she answered "No;" yet if another name than hers were mentioned, she shook her head, whilst if her own were mentioned, she laughed and nodded approvingly. She sometimes remembered her Christian name, *Adèle*, although she pronounced it very badly. She could never be made to say *fork*, *spoon*, *mirror*, *book*, whether the objects themselves were shown to her, without their names being mentioned, or whether their names alone were mentioned. I succeeded, however, in making her count up to twenty, by calling out the figures, one after the other, in her presence. But if, after she had gone through this exercise, I asked her to go over it again by herself, she got muddled before she had counted ten, and never could count up to twenty entirely by herself. Whenever she exerted herself trying to remember and to articulate words, she constantly exclaimed "Oh, pity!" and complained of a painful sensation in the right side of her throat. just as the woman, Marie K——, whose history I have already related. She read almost all day a religious book which the sister had given her, but I discovered that she almost always read the same chapter and the same page; and yet she pretended that she understood well what she read.

As is the case with nearly all aphasic persons, she knew the use of the objects which she could not call by name. Thus, when I put a spoon in her left hand, she lifted it to her mouth, and when I showed her a mirror, she held it before her, and looked at her image in it, laughing. I need not add that the movements of her tongue and lips were normal. She knew *ecarté*, and I played several games with her. I will not say that she played well, but she never, at least, played the wrong colour; she knew well her trumps, and when I pretended to cheat she found it out, and protested, laughing. After all, she spent a whole year in my ward, and although physicians and students, the sisters, the servants, and the other patients kindly tried to teach her, she went from here to the Salpêtrière in nearly the same state as when she was seized, three years previously.[1]

[1] This patient subsequently died in the Salpêtrière Hospital, of her heart disease, on the 19th of July, 1864—that is, about eight months after her removal from the Hôtel-Dieu. The *post-mortem* examination was very carefully made, in presence of Dr. Charcot and M. Broca, twenty-three hours after death, the temperature being very high at the time, and *no lesion of the third*

The next case, which is the analogue of the preceding, is more interesting, from the patient being a very intelligent and educated man.

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left frontal convolution was found. The case was communicated to the members of the Biological Society of Paris, in December, 1864, by M. C. Bouchard, and lately published in the *Gazette Médicale de Paris* (No. 31, August 5, 1865, p. 489), from which we extract the following particulars :—

During her residence at the Salpêtrière, Adèle Anselin had only three sets of words at command. Whenever she wanted to draw anybody's attention to herself, she exclaimed "Mamma, mamma!" When she was asked a question she invariably answered "Cannot say;" and if pressed for an answer, called out "Oh, pity!" She could repeat, however, although very indistinctly, her own name, Adèle, when uttered in her presence. When asked to tell the number of several things placed before her, she counted 1, 2, 3, up to the exact number, if below fifteen, for she never could go beyond. She retained to a greater degree the faculty of expressing her ideas in writing than in speaking. She wrote of her own accord, and with her left hand, a few short sentences like the following: "Sir, I thank you for all your kindness," signing "Adèle Anselin," and adding the name of her former lover. She could also write a few words under dictation, or words which she had just read herself, but she got rapidly tired, left out letters, substituted some for others, noticed at first her mistakes, erased the incorrect words, but did not do better on trying again, and at last merely scribbled with her pen unmeaning characters. Her memory was markedly impaired but retained ideas, which were illustrated by drawings, better than when merely conveyed in printed words. During the eight months which she spent at the Salpêtrière, no modification took place either in her intellectual faculties or in her aptitude for expressing her ideas.

Autopsy.—Festooned excrescences of the sigmoid valves of the aorta; a few atheromatous (non-ulcerated) patches of this artery at its origin; considerable constriction and insufficiency of the mitral orifice. Heart voluminous, dilated, everywhere adherent to the parietal layer of the pericardium. Pericardial sac completely gone, through adherence of both pericardial layers. Depressed cicatrices on the surface of the spleen and the kidneys. In the spleen, in particular, yellow dense striæ, the remains of old obstructions seen in the contracted tissue. On opening the skull, an enormous quantity of sanguineous serosity escaped. The pia-mater was extremely congested. The cerebral arteries were neither calcified nor atheromatous, and contained no concretions. The middle cerebral arteries were particularly examined, and found perfectly pervious. The brain was soft throughout, especially the left hemisphere. The pia-mater, after the blood had been allowed to escape from its vessels, was very thin, but although it was not adherent to the convolutions, the softness of the latter made it very difficult to remove the membranes without injuring them a little. More care was required in removing the membrane on the left than on the right side. The convolutions, when exposed, were of a uniform pink tint, and patches of red points could be seen on them at intervals, as in cases of capillary hæmorrhage. There was one patch, in particular, about the middle of the third left frontal convolution, and another at the lowest part of the left posterior frontal convolution—that is to say, quite close to the third frontal convolution. There were, besides, disseminated and superficial patches of yellow softening on both hemispheres. One of these was situated on the left side, at the point of union of the posterior marginal convolution and the second parietal convolution. Another was seated at the lowest portion of the *insula;* and, moreover, the posterior convolution of the insula was atrophied. On the right side, a yellow patch was also seen on the lowest part of the posterior frontal convolution, extending into the fissure of Sylvius, and a little over the root of the third frontal convolution. The only lesion found in the central parts was yellow softening of the upper and

Mr. T——, a Russian functionary, is nearly sixty years old. He has always enjoyed good health, in spite of fatiguing and assiduous office work. He has never had heart disease. Last year, in the spring, he was seized with paralysis on the *right side*, without loss of consciousness, but with complete loss of speech. From the very beginning his friends were struck with this important fact that his eyes expressed intelligence, that the gestures which he made with his left hand showed great lucidity of mind, and yet that the most direct and pressing questions could only elicit *yes* or *no* from him. For a few months he remained in the same condition, but after that time he could get up, and the paralysis of the arm and leg improved so much that he could walk pretty well, and use his right hand in dressing, but he did not recover his speech.

I saw him in the autumn of 1862, with Dr. Galinzowski, and again on July the 2nd, 1863, on his return from Nice. With the exception of attacks of hepatic colic, which were very severe, but fortunately rare, his general health was good; his aspect was good, he had got stout, and the movements of his arm and leg were embarrassed to a slight extent only. His tongue moved rapidly and freely, and could execute all the movements necessary for articulation, and yet he could not speak. It sometimes

anterior portion of the intra-ventricular nucleus of the left corpus striatum. A consequence of this lesion, showing it to be of old date, was seen in the atrophy of the left half of the pons varolii, and in the very marked atrophy and yellowish-grey discoloration of the left anterior pyramid. On account of this atrophy, the left olivary body looked apparently hypertrophied. Sections of the most seemingly diseased portions of the third left frontal convolution were found normal, when examined under the microscope, so that a proof was thus obtained that the apparent alteration depended solely on congestion, which had occurred in the last hours of life, and on cadaveric imbibition. The nerve-cells were perfectly normal; there was no predominance of the connective tissue, no copora amylacea, no granular bodies, and no granules of hæmatine; the capillaries were healthy, except a very few, which were slightly atheromatous.

M. Bouchard made the following comments on this case:—It does not, he said, confirm M. Broca's theory since the third left frontal convolution was not damaged. It neither adds to, nor subtracts from, Dr. Dax's theory, since both hemispheres were diseased. It may, even in strictness, be regarded as confirming Professor Bouilland's doctrine, since the root of the third frontal convolution was altered, and, *à fortiori*, if, with Professor Trousseau, the name of anterior lobe be given to all the portion of the brain in front of the furrow of Rolando. The aphasia cannot in this case be ascribed to disease of the left olivary body, because the prominence of this organ was only relative, in consequence of the atrophy of the pyramid. The co-existence of various patches of softening in the brain, of traces of embolism in the kidneys and spleen, and of disease of the left heart, render very probable the opinion suggested by Professor Trousseau that the cerebral affection had been the result of an embolon detached from the diseased aortic valves. As the arteries were not found obliterated, however, the obstruction must have been in the capillaries. Lastly, this case confirms the opinion of those who believe that the co-existence of a disease of the heart with right hemiplegia and aphasia, is an important sign for the diagnosis between hæmorrhage into, and softening of, the brain.—Ed.]

happened, however (as in all cases of Aphasia), that he said a word very distinctly and to the point, but was unable to repeat it, when asked to do so, however pressingly.

A remarkable circumstance in Mr. T.'s case is this:—He belongs to the highest circles in St. Petersburg, and speaks French like a Parisian; yet, since his illness, he does not speak a word of French. When I question him, he understands me perfectly, but he always answers with a Russian word. On my telling him, in fun, that he is not polite, because I don't understand Russian, he smiles and says *da*, a Russian word which means *yes;* but he is unable also to construct even a part of a sentence in his own tongue. He, nevertheless, gives signs of intelligence, which are rather curious. His ordinary medical attendant, Dr. Galinzowski, is a Pole, and one can understand that, whilst the present cruel war between Poland and Russia is carried on, the doctor and his patient's friends should not agree.

On one occasion, Dr. Galinzowski was speaking of a terrible engagement which had been fought, near a small village, in his own province in Poland, when M. T——, who had seemed to take part in the discussion, as shown by his animated looks and his agitation, got up, and going to a map, pointed with his finger, after a prolonged search, to the locality which was being discussed; and it turned out that he was right.

He plays whist every day with his daughter, or those of his friends who come to visit him. He plays as well as he ever did; he counts his points well, questions by signs those of his adversaries, and if one of them counts too many honours, he notices it, and by a gesture insists on correcting the error.

These are, certainly, proofs of intelligence and memory; and yet Mr. T. has forgotten the names of things. When asked what a spoon is, he makes a movement showing the use of a spoon; but when asked its name, he does not know it either in Russian or French, and this does not proceed from inability to articulate, but is due to actual forgetfulness. There is no doubt on this point, because when asked if he remembers the name of a spoon which is shown to him, he shakes his head although he says yes, and shrugs his shoulder, at the same time, in a manner expressive of the grief which he feels at his want of memory. When told that it is a pencil or a fork, he shakes his head, although he still says yes; but when the spoon is called by its proper name, he nods approvingly, thus affording the proof that he has forgotten the word, and only remembers it when it is mentioned in his presence.

In August, 1863, a lady came to consult me with her son, aged twenty-five. Four years previously, this young man had, for several days, complained of headache, when he suddenly called out to his mother one morning, "*Oh! I feel something extra-*

ordinary inside me." These were the last words he spoke: his right arm and leg became numb, and after a few hours, the hemiplegia was complete. After a short period he regained some power of moving first his leg, and then his arm; but when he came to me, he still walked with difficulty, and could only use his hand for very rough purposes. The Aphasia, however, which had from the first day been complete, had not diminished. He could articulate two words only:—*No*, and *mamma*. "What's your name?"—"Mamma." "What's your age?"—"Mamma, no." He yet knew that he did not answer as he ought. He had taught himself to write with the left hand, but had not got beyond signing his own name, Henri Guénier. He wrote it very legibly on a piece of paper which I gave him. "Since you write your name," I then told him, "say Guénier." He made an effort, and said "*Mamma*." "Say Henri." He replied, "*No, mamma*." "Well, write *mamma*." He wrote *Guénier*. "Write *no;* he wrote again, *Guénier*. However much I pressed him, I could obtain nothing more. His mother informed me that he played a pretty good game at cards or dominoes. He used to be very fond of reading, and often took up books, which he seemed to read with intelligence; but his mother had noticed that he put the book away after a few minutes, as if he found no interest in it; and yet she took care that the books about him were of easy comprehension as well as amusing. His face looked intelligent as it does in most cases of Aphasia; but as he had perfect health and had no headache, and as his sight was excellent, his intellect must have been somehow impaired, since he found no charms in books which would formerly have amused him.

I observed another case of the kind, with Drs. Campbell and Blondeau. An English banker, a resident in Paris, aged 42, robust and stout, fond of good living, and of a lively, cheerful temperament, went out as usual in his carriage, on the 9th of April, 1863. He was returning home to breakfast, about eleven o'clock, when, on stepping out of his carriage, he dropped down without losing consciousness. His whole right side was paralyzed, and the paralysis had probably begun, without his perceiving it, during the latter portion of his drive. The porter carried him into his lodge, and Drs. Campbell and Blondeau were fetched, and arrived at the same moment with one of my colleagues in the faculty. There was complete right hemiplegia, sensibility being almost completely abolished, whilst the most violent irritation could not excite any movement. The patient tried to speak, but could not articulate a single word, and scarcely succeeded in uttering a few grunts; yet his eyes were full of intelligence, and he seemed to understand the questions that were put to him. There had been no coma, no stertor.

Drs. Campbell and Blondeau refused to bleed the patient, as was advised by the third physician. They recommended that he should lie down with his head propped up, and merely prescribed acidulated and slightly laxative drinks, trusting more to hygienic means than to active treatment for warding off danger, although they were aware that the left side of the brain was irretrievably damaged. I saw the patient in the evening, in consultation with these two gentlemen, and thoroughly approved their prudence, feeling confident that the patient's life would have been gravely compromised by bleeding and violent purgation, by blisters and those numerous remedies which are used against a deep lesion, which has, as a rule, occurred when the physician is called upon to interfere.

On the two following days fever was lighted up, and symptoms of pulmonary congestion showed themselves, which gave us great anxiety; but a little calomel and musk soon got rid of these, and we could entertain the hope that the patient's life would be saved. The pulse became quiet again, and respiration natural; light food was taken well, and on the twelfth day the patient was made to get up and to sit in an easy chair. Since then, up to the present time, the amelioration has gradually increased, but the faculty of speech is almost nil, although the patient can walk by leaning on a friend's arm, and can use his hand to some extent. For more than three months he has only been able to say a few words, devoid of meaning, and always the same; on one occasion, however, a fortnight after the attack, he distinctly said "*My dear*" to his wife. But we never could make him repeat those two words. At present he can say a few words, but very few, and they are not always used rightly. Eight months after his seizure, he had in December, 1863, an attack of eclampsia, and another again in February, 1864, and these attacks will probably recur again.

I now pass on, gentlemen, to the case of a man who died in my wards, after having presented, during life, the most characteristic symptoms of Aphasia, and whose brain was examined after death with the utmost care, in Dr. Broca's presence.

The patient was 60 years of age. He had been, at first, under the care of Dr. Vigla, who transferred him to me. His intellect seemed to be impaired; he had been paralysed for several months, and although he looked as if he understood, when he was spoken to repeatedly, he never answered anything beyond "*Oh! mad.*" General sensibility was normal, and when he was pinched hard, he exclaimed in a more decided tone: "*Oh! mad,*" and shook his head to show that he was annoyed. He died a few weeks after admission. As this was an important case, which might confirm or upset the theory concerning the localization of intellectual faculties, I requested Dr. Broca to be

present at the post-mortem examination. The brain was removed with care, and there was found on the left side yellow softening of the lower marginal convolution, of the lower portion of the transverse parietal convolution, and of the convolutions of the insula. At first sight, the frontal lobe seemed to have escaped; but on drawing away the edges of the Sylvian fissure, the softening was seen to extend, from the convolutions of the insula, to the lower portion of the transverse frontal convolution, and moreover, that the third frontal convolution was itself softened in its posterior portion, that is, in the part nearest the sulcus of Rolando.

The morbid specimen was exhibited by M. Dumontpallier, my then clinical assistant, to the members of the Biological Society, at their meeting on the 28th of March, 1863; and, at Dr. Broca's request, it was afterwards placed in the Dupuytren Museum, where it can now be seen, I believe, so that anyone may ascertain for himself that the lesion was really seated in a portion of the sphenoido-temporal lobe, and in the third frontal convolution. This case, therefore, supports Dr. Broca's views. M. Dumontpallier showed also, at the meeting, that the left middle cerebral artery was obliterated by a fibrinous clot, which might have been ascribed to Embolism (as the walls of the artery were healthy), if the examination of the heart had ascertained this point. None of the cerebral arteries were atheromatous, however, the right middle cerebral artery was pervious, and there was no trace of defective nutrition in the right hemisphere of the brain.

Now that I have related to you this interesting case, I will pass on to the anatomical questions bearing on Aphasia, and discuss what has been written on the subject.

The physiological conditions of Aphasia have long ago been observed. I need only mention the following passage of Pliny, in which the learned naturalist observes that nothing in Man is so fragile as Memory: "Illness, falls, a mere fright, impair it partially, or destroy it completely. A man, struck by a stone, forgot the letters of the alphabet; another, who had fallen from a very high roof, no longer recognized his mother or his friends; a third, after a severe illness, forgot that he possessed slaves; and Messala Corvinus, the orator, forgot his own name."[1] Schenkius,[2] who lived at the end of the 16th century, noticed that, in some cerebral affections, although the tongue was not in the least paralyzed, the patients could not speak, because they had lost their memory: "Observatum a me est plurimos,

[1] Pliny. Natural History, book vii., sec. 24.
[2] Joan Schenkii, *Obs. Med.*, lib. vii. in fol., p. 180. Lugduni, 1585.

post apoplexiam aut lethargum, aut similes magnos capitis morbos, etiam non præsente linguæ paralysi, loqui non posse, quod memoriæ facultate extinctâ, verba proferenda non occurrant."

In 1820, the illustrious Lordat,[1] who became aphasic eight years afterwards, ascribed this affection, which he termed *alalia*, not to paralysis of the tongue, but to a defect of co-ordination of the muscles which are used in the act of speaking. But Lordat did not attempt to specify the part of the Brain an injury to which might cause loss of speech. Gall conjectured that the faculty of Articulate Language was located in the anterior lobes of the brain. Dr. Bouillaud, who studied and adopted some of Gall's theories (within restricted limits, it is true), was led, by clinical observation, to locate this faculty in the frontal lobes. "The anterior lobes of the brain," said he, in the year 1825, at page 284 of his *Treatise on Encephalitis*, are the organs "for the formation and recollection of words, or the principal signs which represent our ideas." Whilst, in the next page, he emphatically declares that "the anterior portion of the brain is the organ of articulate language."

In 1836, Dr. Marc Dax, of Sommières (Gard.), read at the medical congress of Montpellier, a very interesting and original essay, in which he attempted to specify, with greater precision than Dr. Bouillaud had done, the part of the Brain which he regarded as the seat of the manifestations of thought by speech. So far back as the year 1800, he had noticed that patients suffering from Aphasia, when paralyzed at the same time, were paralyzed on the *right* side, and that consequently the anatomical lesion was seated in the left hemisphere. When once his attention had been called to this curious point of pathological physiology, he ascertained clinically that, when there was loss of memory of words, the lesion was always seated on the left; and he added that he had never met with this affection in cases of cerebral disease exclusively limited to the right hemisphere. The title of his essay, besides, sums up his views: *Lesions of the left half of the Brain coinciding with the loss of memory of the signs of thought.*[2]

Thus, gentlemen, we see Dr. Bouillaud demonstrating by facts what Gall had obscurely seen; namely, that the material condition on which depends the memory of the principal signs of thought is the integrity of the anterior lobe of the Brain; whilst Dr. Marc Dax localizes this faculty in the left hemisphere exclusively.

[1] Rev. pér. de la Société de Méd. de Paris. Décembre, 1820, p. 317.

[2] This essay has been lately published in the *Gazette Hebdomadaire*, Paris, 28 Avril, 1865, No. 17, p. 259; and an abstract of Dr. G. Dax's paper is also given in the same number.—Ed.]

In the beginning of 1863, Dr. G. Dax, following in his father's steps, sent to the Academy of Medicine a memoir, in which he tries to prove that, in Aphasia, the lesion is not only invariably seated in the left hemisphere, but in the anterior and outer portion of the *middle lobe* of that hemisphere. This statement, made in 1863, differs very little, as you may see, from what Dr. Broca has lately shown; for the spot in which Dr. G. Dax locates the lesion is evidently very close to the *insula of Reil*, and consequently to the posterior portion of the frontal lobe.

But Dr. Bouillaud, whilst inclining to the opinion that Aphasia is, in most cases, due to a lesion of the anterior portion of the anterior lobes of the Brain, admitted also that the same morbid phenomenon could be produced by disease of the posterior portion of these lobes. In the memoirs read by Dr. Bouillaud at the Academy of Medicine on the 22nd of February and the 7th of March, 1848, these points are well established.

In 1856, Dr. Marcé tried to show that there is a co-ordinating principle for the acts of speaking and writing (*Memoirs of the Biological Society*); and that, in a certain number of cases, the faculty of articulate language can be lost independently of the faculty of language as expressed by writing, or *vice versâ*; but he denied that a special portion of the Brain could be assigned as the seat of this co-ordinating principle.

In 1861, at the Anthropological Society of Paris, an important discussion arose concerning the localization of the cerebral functions, in which Drs. Gratiolet, Auburtin, and Broca took the most prominent part. Dr. Gratiolet maintained that all attempts at localization which had been made up to that time, had no basis. Dr. Auburtin affirmed the reverse proposition, citing Dr. Bouillaud's researches, several cases related in Dr. Rostan's works and in Lallemand's letters, and concluding that the anterior lobes of the Brain were the seat of the co-ordinating faculty of speech.

The authority of Dr. Gratiolet who had studied, specially and deeply, the anatomy and physiology of the nervous system, on the one hand, and the facts quoted by Dr. Auburtin from justly-esteemed works, on the other hand, *rendered the solution of the point at issue difficult.* Was the Brain to be regarded as a great whole, all the parts of which, as well as their faculties, are mutually dependent? or should it be divided into departments, and the department of each faculty ascertained? M. Broca hesitated like the rest; and yet he was one of the first to bring forward cases lending great support to the theory of cerebral localizations. A few weeks after the discussion at the Anthropological Society, a man, named Leborgne, 51 years old, and who had lost speech for twenty years, was transferred to his care, in Bicêtre. The case is related in detail in the *Bulletins de la*

Société Anatomique, August, 1861; but the following is a summary written by M. Broca himself for the Proceedings of the Anthropological Society.

The patient was admitted into Bicêtre twenty-one years ago. Shortly before then, he had lost the power of speech, and could utter one syllable only, which he usually repeated twice in succession. Whatever question was asked him, he always answered: *tan, tan*, accompanying his answers with very varied and expressive gestures. Throughout the asylum he was therefore known by the name of *Tan*.

At the time of his admission he was intelligent and could use all his limbs perfectly. After ten years, he gradually lost the power of moving his right arm, and next his right leg; so that, for the last six or seven years, he has been constantly confined to his bed. For some time past, his sight has been noticed to grow weaker; and, lastly, those who were frequently about him observed that his intelligence had failed a good deal within the last few years. He was transferred to M. Broca's care on account of a diffuse gangrenous inflammation of the cellular tissue of the whole lower extremity, on the right or paralysed side, extending from the instep to the buttock. The case of this poor fellow, who was unable to speak and write, was somewhat difficult to study. It was ascertained, however, that common sensibility was nowhere impaired; that the left arm and leg moved in obedience to the will; that the muscles of the face and tongue were not paralysed; and that this last organ moved very freely. There was no doubt, according to M. Broca, that the "*patient's intellect was deeply damaged*," but that more of it was retained than is required for speech. Besides, Tan was perfectly intelligent for sixteen or seventeen years, although he had been unable to speak for twenty-one years. He died on the 17th of April, 1861.

At the *post-mortem* examination, the dura mater was found thickened and vascular, lined on its inner aspect by a thick pseudo-membrane; the pia-mater was thickened and opaque over the anterior lobes, to which it was adherent, especially on the left side. The frontal lobe of the left hemisphere was softened in the greater part of its extent; the convolutions of the orbital lobule, although atrophied, had preserved their shape; most of the other frontal convolutions were destroyed. From this destruction of the cerebral tissue, there had resulted a large cavity, of the size of a hen's egg, filled with serosity. The softening extended backwards to the ascending portion of the parietal lobe, downwards to the marginal portion of the temporo-sphenoidal lobe, and inwards to the lobule of the insula and the extra-ventricular nucleus of the corpus striatum. To the disorganization of this last part must be ascribed the motor paralysis of the upper and lower limbs, on the right side. The oldest and

most extensive lesions, however, were found in the middle portion of the frontal lobe of the left hemisphere. The neighbouring portions had softened very gradually only, and it may be considered a certainty that, for a very long period, the convolutions of the frontal lobe were alone affected.

This period probably comprised the eleven years which preceded the paralysis of the right arm, and during which the patient's intellect was unimpaired, and speech alone was lost. It was, therefore, allowable to ascribe, in this case, the loss of speech to the disorganization of the frontal lobe, particularly when Professor Bouillaud's views were kept in mind.

M. Broca's second case, however, seemed to point to a very limited part as the seat of the faculty of articulate language. A man, aged eighty-four, had been eight years previously admitted into Bicêtre, on account of senile debility. He was not paralysed at that time; his senses, his intellect were perfect. In April, 1860,—that is to say, when he was eighty-three years old,—Lelong was seized with apoplexy, whilst going down a staircase. A few days afterwards he left the infirmary, having never been paralysed, but having suddenly and completely lost the power of speech. He could only articulate a few words with difficuty; his gait was somewhat uncertain, but he was not lame; his intellect did not seem to have been appreciably impaired; he understood what was said to him, and his small stock of words, accompanied by an *expressive pantomime*, enabled him to make himself understood in his turn by the people who lived habitually with him.

On the 27th of October, 1861, Lelong was admitted into the infirmary, under M. Broca, on account of a fracture of the neck of the femur, on the left side. He was *not paralysed* of motion or sensation; his tongue moved freely in all directions, he could swallow well, all his senses were normal, and his intellect was unimpaired. Yet, when questions were put to him, he only answered by signs, uttering at the same time one or two syllables suddenly and with a certain degree of effort. He could only say, *yes, no, three,* and *always;* when he was asked his name, he replied *Lelo* instead of Lelong which was his real name. He said *yes* and *no,* at proper times; but he made use of the word *three* in order to express any number, although he knew well that the word did not always convey his meaning, and corrected the mistake which he made in speaking by holding out the proper number of fingers. He could tell the time by a watch, and had retained the notion of units and tens. He had not lost his memory, and on one occasion only, according to M. Broca, was his memory at fault, when he was asked how long ago he had lost the power of speech.

M. Broca sums up the history of this case, which is published *in extenso* in the *Bulletins de la Société Anatomique* (November,

1861), by affirming :—1. that Lelong understood all that was said to him; 2. that he used with judgment the four words of his vocabulary; 3. that he was of sane mind; 4. that he knew written numeration, and at least the value of the first two orders of units; 5. that he had lost neither the general faculty of language nor the power of moving the muscles which are used in the production of sound and in articulation, and that he had consequently lost the faculty of articulate language alone. He was, therefore, aphasic. M. Broca does not, however, in his conclusions, call attention to the fact that Lelong who knew how to write, and whose hand was not paralysed, could not *guide* his hand so as to form letters; although the fact itself is mentioned in the detailed and complete history of the case, as given by M. Broca.

The patient died on November the 8th, 1861, twelve days only after his fall, from the consequences of the fracture of his femur, and without having suffered from any cerebral complication. The *post-mortem* examination was made with the greatest care. The right hemisphere was found healthy throughout, as well as the cerebellum, the pons varolii, and the medulla oblongata. In the left hemisphere, the thalamus opticus, the fornix, corpus callosum, corpus striatum, the occipital and parietal lobes, the lobule of the insula, and the orbital convolutions which form the inferior layer of the frontal lobe, were healthy. It was thought, however, that at the point of union of the anterior extremity of the ventricular nucleus of the corpus striatum with the medullary substance of the frontal lobe, the consistency of the brain substance was slightly diminished; but this lesion, M. Broca adds, if it can be regarded as such, was totally independent of the principal one, and separated from it by a considerable thickness of healthy tissue.

Before describing, however, the lesion which was found, in this case, to be perfectly limited to a portion of the posterior third of the second and third left frontal convolutions, it will be necessary to give a brief description of the arrangement and relations of the cerebral organs which shall have to be mentioned.

The sulcus of Rolando separates the frontal from the parietal lobe, running obliquely from above downwards along the outer surface of the hemisphere, and beginning at the median fissure between the two hemispheres, and ending in the Sylvian fissure. It is limited, anteriorly, by the transverse frontal convolution, posteriorly, by the transverse parietal convolution. The anterior or frontal lobe comprises, therefore, laterally, all that portion of the hemisphere which is situated in front of the sulcus of Rolando, and inferiorly, all that portion which is in front of the Sylvian fissure. The lower portion of the frontal lobe consists of the

orbital convolutions, whilst its upper and lateral portions are constituted by the frontal convolutions properly so called. These are three in number: an upper or first frontal convolution, a middle or second convolution, and a lower or third frontal convolution. They are all directed from before backwards, and terminate after a more or less tortuous course, in the transverse frontal convolution of which they seem to be the ramifications. The third frontal convolution is free in its upper half, and separated from the temporo-sphenoidal lobe by the Sylvian fissure of which it forms the upper margin. It is on account of this relation that the third frontal convolution is sometimes termed the *upper* marginal convolution, whilst the name *lower* marginal convolution is restricted to the first temporo-sphenoidal convolution. When the two marginal convolutions, the upper and lower, are drawn away from the Sylvian fissure, there is seen a large and slightly prominent eminence from the summit of which proceed five small simple convolutions, or rather five rectilinear folds radiating in a fan-like manner. This eminence is the *lobule of the insula*, which covers the extra ventricular nucleus of the corpus striatum, and which, rising from the bottom of the Sylvian fissure, is structurally continuous by its cortical layer with the deepest portion of the two marginal convolutions. The result of these structural relations is, that a lesion which extends by continuity from the frontal to the temporo-sphenoidal lobe or the reverse, must necessarily pass through the lobule of the insula and then affect the extra ventricular nucleus of the corpus striatum.

These anatomical details, which I chiefly borrow from M. Broca's memoir, easily account for certain very limited lesions, such as those found in Lelong's case. In his case, indeed, the posterior third of the second and third left frontal convolutions was alone destroyed, over a space of about 15 or 18 millimetres. The transverse frontal convolution was normal, whilst inferiorly, the lesion extended as far as the lobule of the insula, but without involving it. The result of this loss of substance was a cavity full of serosity and closed externally by the pia-mater. The walls of this cavity were firm, and on them were small spots of an orange-yellow colour, probably of blood-origin, and afterwards proved to be so by the microscope. This was, therefore, an old hæmorrhagic cyst, and the patient, as you know, had suddenly lost his speech since a fit of apoplexy which he had eighteen months before he died.

This case proves, therefore, that when there is no other brain lesion than a loss of substance of the posterior third of the second and third left frontal convolutions, there may solely exist, either as a coincidence, or as a consequence, loss of the faculty of articulate language.

Thus, gentlemen, we have Professor Bouillaud placing the

organ of the manifestations of thought by speech in the two anterior lobes of the Brain; Dr. Marc Dax locating it in the left hemisphere exclusively; and Dr. G. Dax, at the point of union of the middle with the frontal lobe of the left hemisphere; whilst M. Broca points to a more definite spot, and although he did not probably know of Dr. Marc Dax's essay, and certainly not of Dr. G. Dax's researches, he, like them, points to the posterior portion of the left frontal convolution as the seat of the faculty of speech.

If there were on record several cases exactly similar to the one which I have just related, we should be compelled to admit a relation of cause and effect between the seat of the anatomical lesion and the loss of speech. Dr. Charcot, in 1862 and 1863, exhibited to the members of the Biological Society several brains removed from old women who had died in the Salpêtrière, and who had, for a variable period during life, suffered from loss of speech. In most instances, the lesion was complex as in M. Broca's first case. Thus, there had generally been paralysis as well as aphasia, and the second and third frontal convolutions were not the only spots where there was softening or hæmorrhage, but the lobule of the insula and the temporo-sphenoidal lobe were also the seats of an anatomical lesion which had probably occurred simultaneously everywhere, or which had resulted from the extension of the softening by continuity of tissue.

The softening was frequently of an amber-yellow colour, and varied much in degree, both as regards depth and extent. A very remarkable fact, however, was, that in the first ten or twelve cases related by Dr. Charcot, the third frontal convolution was disorganized at its posterior extremity, thus lending considerable support to M. Broca's theory.

Subsequently, however, Dr. Charcot was the first to communicate to the members of the Biological Society a case of Aphasia without damage to the third frontal convolution.

This case has been published in the *Gazette Hebdomadaire*, and M. Broca, who assisted Dr. Charcot at the *post-mortem* examination, has fully acknowledged its value, and that it invalidated the anatomical law which he had laid down. The patient was forty-seven years of age, and had become hemiplegic and aphasic since an apoplectic fit which she had had eight months previously.

Intellect and memory seemed to be preserved, for, during her stay in the infirmary, she recognized patients whom she had formerly seen in the Salpêtrière. But her power of articulate language "was restricted to the utterance of the monosyllable *ta* which she habitually repeated, with very great rapidity and distinctly, four or five times in succession (*ta ta ta ta*) whenever

she attempted to answer a question or to communicate her own ideas. The tongue moved freely and in all directions."

An examination of the Brain, after death, showed that there was softening: "1st, of the so-called lower marginal convolution in all its extent, and of a portion only of the second temporal convolution, of the temporal lobe; 2nd, of the lower extremity and the whole of the two posterior convolutions of the insula of Reil. In depth, the softening extended in the direction of the corpus striatum; the whole of the extra-ventricular nucleus and the posterior half of the intra-ventricular nucleus were also softened. The thalamus opticus was normal."

The transverse parietal and transverse frontal convolutions, the three antero-posterior frontal convolutions, known by the names of first, second, and third frontal convolutions, were examined thoroughly, one after another, with the greatest care, in M. Broca's presence. To the naked eye, they did not seem to have undergone any change of size, colour, and consistency. Lastly, thin sections of several portions of the third frontal convolution were examined under the microscope, and the nerve-tissue was found unchanged; here and there only, two or three granular bodies were seen on each microscopic preparation. But neither Dr. Charcot nor M. Broca himself regarded the presence, in such small numbers, of these granular bodies as a sign of organic lesion, as they can be met with in nerve-tissue without any co-existing functional disturbance. Let it be kept in mind, besides, that both Dr. Charcot and M. Broca were anxious to determine the presence of a pathological lesion; and if they declare that none was to be found, we must certainly conclude with them, that the faculty of articulate language may be destroyed without there necessarily being an appreciable organic alteration of the third left frontal convolution.

The following case, observed by Dr. Vulpian, equally proves, in my opinion, that Aphasia may be caused, as Dr. Marc Dax had conjectured, by lesions of the left hemisphere independent of the frontal lobe.

"A woman, aged seventy-three, is transferred to the infirmary at the Salpêtrière, on the 15th of December, 1863, because for a few days past she had been observed to grow markedly weaker.

"When first seen by Dr. Vulpian, she had no fever and seemed to suffer from no thoracic or abdominal complaint; but she was not able to speak, and all the attempts to make her say a single word were fruitless. She seemed to understand what was said to her, tried to answer, but on rare occasions only succeeded in stuttering unintelligibly; in general, she uttered no sound at all. She had no paralysis of the limbs, face, or tongue; she squeezed pretty hard and equally well with both hands; she walked without

help, but slowly, and taking short steps, without dragging either leg. She did nothing extravagant.

"She was watched day after day, and for the first ten days her condition did not change. Her intellect was evidently of a low grade, although she correctly nodded or shook her head, according as she meant yes or no. 'One morning I found her in tears' (says Dr. Vulpian), 'and as soon as she saw me she went through a pretty expressive pantomime which suggested to me the idea that she might have been beaten; and I indeed learnt that a neighbour of hers, who was delirious, had got up during the night and struck her repeatedly. I must add, that her pantomime, although expressive, was not so clear as would have been that of a perfectly intelligent person.'

"On one occasion only, she said, '*Yes yes, sir,*' but could not be made to repeat the same words on the following day. Ten days after her admission, after looking more prostrated than usual during the night, she was found one morning partially paralyzed on the right side; on the next day, the paralysis had become complete. A few days afterwards, the face deviated a little (the left commissure being pulled a little towards the ear); there was some tendency to contraction of the paralyzed arm. No appreciable change in the patient's intellectual condition was noticed with regard to speech, although she no longer uttered the sounds which she did before. A month after her admission, she had Pneumonia on the right side, of which she died at the end of six days.

"The persons who had been in the same part of the asylum with her, declared that she was not able to speak when she was *first admitted into the Salpêtrière,* and one of them even went so far as to assert that the patient had lost her speech *three years before she was transferred to the Infirmary.* I had felt no hesitation, therefore, in regarding this case as a typical one of Aphasia, which was all the more remarkable from there having been no paralysis at first, and from right hemiplegia having occurred towards the close.

"Dissection, however, did not disclose the lesion which I fully expected. I found a broad patch of softening, of apparently recent date, in the posterior half of the white supra-ventricular nucleus of the left cerebral hemisphere, and no trace of disease in the frontal or other convolutions. Old lesions, slight in degree, lacunæ, were seen in the corpus striatum and the thalamus opticus on the same side, and an analogous lesion, of still smaller extent but of as old a date, in the right corpus striatum.

"This case, therefore, was apparently an exceptional one, and by ascribing a somewhat old date to the softening (a central portion of it looked, certainly, a little older than the rest) the Aphasia could be referred to this lesion. So that this would

have been a case of Aphasia produced by a lesion of the posterior part of the hemisphere. Luckily I found some notes about this patient who, six months previously, had been under my care for nine days. At that time she spoke, and could articulate any word. She could ask for what she wanted, and even talk a little with other patients. It was true that she spoke very little, with some difficulty in finding words. Articulation was slow when a sentence had to be spoken, whilst, on the contrary, the following words came out explosively, as it were: *Yes, sir, yes. No, sir, no.* She never said *yes* or *no* in any other way, except when repeatedly pressed. She had no paralysis of the face, eyes, tongue, or limbs, but there was already some weakness of the lower limbs. She told me at that time that her speech had become embarrassed three months previously, after repeated attacks of giddiness several days in succession. Since then, she occasionally felt giddy, and her speech became more embarrassed. These notes, therefore, to some extent modify the conclusions which might be drawn from the case.

"Both middle cerebral arteries were very atheromatous; but whilst the right artery was still pervious to the blood, the left one was completely or almost completely plugged up (the plugging seemed to be complete in one point at least) in two places, separated by an interval of about one centimetre from one another, owing partly to the atheromatous thickening of its walls and partly to an indurated fibrinous deposit of manifestly old date. This deposit seemed to have been the result of thrombosis rather than of embolism. It is probable that the plugging of the vessel was the cause of the first symptoms. Circulation was considerably impeded on several occasions, but was probably re-established, although incompletely, by collateral channels. I can thus account for the old and partial patches of softening, resulting in the lacunæ, which dissection disclosed in the two corpora striata and in the left optic thalamus, and the somewhat intermittent embarrassment of speech, as well as the weakness of the lower limbs and the failure of intellect. All these symptoms were due to insufficient nutrition of the Brain. The right cerebral hemisphere must have been also imperfectly nourished, although to a less degree than the left hemisphere, since the walls of the left middle cerebral artery were atheromatous.

"When the patient was for a second time admitted under me, she was then suffering from one of the attacks to which she was liable, and during which the impediment in her speech was so exaggerated as to merge into Aphasia. The cerebral softening set in next, doubtless owing to the persistent plugging of a portion of the arterial system, which, until then, had been more or less pervious to the blood."

M. Fernet, house-physician to the hospitals, communicated to

the Biological Society, in March, 1863, a case of complete left hemiplegia, with softening of the right frontal lobe and thrombosis of the middle cerebral artery on the same side.

The patient, a woman aged forty-six, had not been aphasic. The whole of the frontal lobe was in a pulpy condition, and although it is not specified that the third frontal convolution was softened, it was no doubt affected, and M. Fernet has himself informed me that it was. The temporo-sphenoidal lobe and the convolutions of the insula were not involved.

Since this patient was not aphasic, we must conclude that the frontal lobe on the right side may be entirely disorganized, without aphasia following of necessity. Some very severe critics might not be disposed to set much value on this case, because the precise limits of the softening are not given in detail; but M. Fernet, it should be observed, did not draw attention to the question of Aphasia, because, as he said himself, he was not sufficiently acquainted with the subject. This case becomes of great importance, however, when it is placed by the side of the one published a few months later by Dr. Parrot in the *Gazette Hebdomadaire* (July 31, 1863), under the following heading: *Complete Atrophy of the Lobule of the Insula and of the third Convolution of the frontal Lobe, with Retention of Intelligence and of the Faculty of Articulate Language.* The pathological portion of this case does not admit of criticism, and it must be acknowledged that there was really softening of the posterior third of the frontal convolution,—of the very spot, in fact, where M. Broca had located the faculty of Articulate Language.

The following case, which Dr. Charcot has kindly communicated to me, is exactly like Dr. Parrot's.

"Egris-Valentine Thérèse, aged seventy-seven, is admitted into the Salpêtrière on the 21st of December, 1863, on being discharged from La Pitié, where she had been for three months under Dr. Marrotte's care.

"*Intelligence and memory seemed to be remarkably good.* The patient declared that, about three months ago, she was seized with complete paralysis of the left side; she fell down, lost her senses, and remained insensible for nine hours. She was carried to La Pitié, and her speech, which had been impeded at first, soon became natural again. Whilst she was in La Pitié Hospital, her lower limbs and her left arm swelled considerably; and they were still ædematous. The swelling was preceded by diarrhœa which had continued ever since. For the last month the patient had had no control over her bladder or rectum, and a patch of gangrene had formed over the sacrum. *There was no embarrassment of speech, no forgetfulness of, or mistake in, words when speaking.*

"The patient died of pneumonia on January the 3rd, 1863; and at the *post-mortem* examination, the following lesions were

found:—The amount of sub-arachnoid fluid is considerable; there is very extensive yellow softening of the outer surface of the right frontal lobe, with nearly complete atrophy of the convolutions. The parts softened are the anterior marginal convolution, and the second and third frontal convolutions which are completely destroyed, and the posterior part of the lobule of the insula. Microscopical examination shows, in the diseased parts, numerous granular corpuscles, a considerable amount of fatty granules in the intercellular tissue, and atheromatous degeneration of most of the blood-vessels.

"The central parts are healthy—namely, the corpora striata, thalami optici, and lateral ventricles. The right crus cerebri is markedly smaller than the left, and is of a greyish tint. In the interstices between its nervous elements, a certain number of granular corpuscles are found. The pons varolii is flattened on the same side, as well as the anterior pyramid which differs from the left one, both in respect of its smaller size and its greyish tint like that of the crus, and like it owing to the granular bodies. The upper portion of the spinal cord was alone examined, and its left half was smaller than the right, the diminution in size being chiefly due to that of the antero-lateral columns."

You see, gentlemen, that in this case, as in those reported by M. Fernet and Dr. Parrot, the third frontal convolution was seriously damaged; but the lesion was seated on the *right* side, whereas M. Broca maintains that disease of the third convolution of the *left* frontal lobe can alone produce Aphasia.

The cases on which M. Broca's memoir is based—the more numerous ones which I have just quoted, others published by Drs. Vulpian, Charcot, and Perroud—seemed to establish incontestably, not that Aphasia was of necessity produced by a lesion of the third left frontal convolution, since Dr. Charcot's case has done away with that opinion, but at least that it was only produced by a lesion of the *left cerebral hemisphere*, and never by a lesion of the *right*, as Dr. Marc Dax had shown. Indeed, there was not a single authentic case on record of Aphasia with left hemiplegia.

The three cases published by MM. Fernet, Parrot, and Charcot prove that lesions, which on the left side produce Aphasia, do not cause it when seated on the right side. But M. Broca believed that he was in a position to affirm (however bold this opinion might be in a physiological point of view) that the faculty of Articulate Language was structurally dependent on the integrity of the third left frontal convolution. He did not attempt to explain this strange localization, but simply noted the facts which seemed to declare in his favour.

You see, gentlemen, that I have kept back none of M. Broca's arguments, and that I have allowed them to be stretched almost

to the limits of absurdity; for is it possible in physiology to admit that in an organ so exquisitely symmetrical as the Brain, there may be in one of the hemispheres a portion discharging a function which does not appertain to the other hemisphere? Analogy and common sense would protest against such a conclusion, and although, in almost all the cases of Aphasia which have come under my observation, the paralysis (when present) always affected the right side, and I was therefore obliged to admit a lesion of the left hemisphere, I could not accede to M. Broca's strange doctrine. You remember the case of Marcou which I have already related to you, and which proves that Aphasia, in its most characteristic form, may accompany left hemiplegia, and consequently, a lesion of the right hemisphere. M. Broca's doctrine was, therefore, upset by such a case, although it is true that when Aphasia is attended with paralysis, as it most frequently is, the lesion is nearly always on the left side of the brain whilst the loss of motion is on the right side.

I know very well that certain objections may be raised about Marcou's case. It will not be said that he was not affected with Aphasia, but it will be suggested that as there was no autopsy, two lesions might be admitted, one causing left hemiplegia and seated in the right hemisphere, the other producing Aphasia without hemiplegia, and seated in the third left frontal convolution. I admit that, in the absence of an anatomical demonstration to the contrary, this is not impossible; but I must call your attention to the fact that Marcou became aphasic at the very moment when he was seized with left hemiplegia. There must have been, then, two simultaneous lesions—one of the left frontal convolution, and the other of the right hemisphere. Now such cases do pretty frequently occur, and I have, on several occasions, shown you multiple apoplectic cysts in the brain of individuals who had died of cerebral hæmorrhage. But such multiple cysts are only found in cases of severe apoplectic attacks, and very rarely in such mild seizures as Marcou's. They are pretty frequently met with, also, as a consequence of falls on the head. Thus, in a case observed by M. Ange Duval, surgeon to the Naval Hospital at Brest, and communicated by M. Broca to the Surgical Society, on the 24th of February, 1864, the patient became aphasic after a fall on the head, and there was found at the same time, in the *right anterior lobe*, a sanguineous cyst with superficial alteration of the orbital convolution, and on the *left* side laceration of the third frontal convolution, which was completely softened. For the present, however, I am justified in regarding Marcou's case as one of *Aphasia with lesion of the right hemisphere;* and I think that the following conclusions may justly be adopted:—

Aphasia is produced in nearly all cases by an injury to the frontal lobes, as Professor Bouillaud has shown.

The lesion, as Dr. Marc Dax has established, is almost exclusively confined to the left hemisphere; whilst its most frequent seat is the posterior part of the third left convolution, as M. Broca was the first to point out.

Now, gentlemen, let us examine the question in another point of view.

If it be easily admitted that Aphasia, when accompanied by paralysis, is due to softening or hæmorrhage, it is difficult to conceive the nature of the lesion when the Aphasia lasts a few minutes or a few hours only, and is not accompanied or preceded either by headache or by paralysis, even of a transitory character. Yet it is still more difficult to deny the existence of a lesion. I grant that this lesion is neither softening nor hæmorrhage, but there must have been some modification in a portion of the brain, and probably in the same part which is deeply damaged in cases of Aphasia attended with paralysis, a modification which is perhaps the analogue of the transitory congestions which we observe in certain exposed parts, or of those deep disturbances of the capillary circulation which sometimes manifest themselves by hyperæmia, sometimes by anæmia, sometimes by the loss or by the exaltation of sensibility.

We are driven to conjectures, as you see, gentlemen. But I wish to call your attention to an important fact which has not passed unnoticed, but which has been too much lost sight of by practitioners—namely, to remains of old lesions which are found in the brain of individuals who, for several months, had had Aphasia without paralysis, and who died of some acute cerebral affection or of some complaint independent of Aphasia. Clinical experience proves, therefore, that there may exist in the brain lesions of sufficient gravity to cause persistent Aphasia without producing paralysis; and it is not impossible that a small hæmorrhage may cause Aphasia of a few hours' duration, in the same manner as we see it produce, in some cases, paralysis of one, two, or three days' duration. How often are the remains of eight or ten successive hæmorrhages found on dissection in the brain of individuals who have only had two or three paralytic strokes? I could not, therefore, affirm that cases of transitory Aphasia (which are not infrequent) are not produced either by a small hæmorrhage or by the softening of a very limited portion of the frontal lobes. This view derives support from the fact that aphasic individuals, who for several months have given no evidence of paralysis, pretty often die after a violent attack of hæmorrhage into, or of softening of, the brain, as in the case of the woman Desteben, which I related to you at the commencement of this Lecture.

The seat of these small hæmorrhages or of this partial softening which only cause temporary paralysis scarcely noticed by

the patient or his friends, is of very great importance. For if lesions of the frontal lobe very often cause the loss of the faculty of manifesting thoughts by speech, writing, and gesture, they possess a very limited influence on the loss of sensation and motion. If we admit with Professor Bouillaud, as I am inclined to do, that this part of the brain is the seat of the faculty in virtue of which thoughts are manifested by speech, writing, and gesture, whilst other parts hold more especially under their dependence the faculties of motility and sensibility, we can better conceive how the frontal lobes may be slightly injured and yet produce no hemiplegia, and how the optic thalamus, corpus striatum, or centrum ovale of Vienssens may be slightly damaged, and yet cause only a small degree of hemiplegia without affecting the faculty of speaking and of writing.

Dr. Auburtin goes further, and proves by clinical cases, some of which came under his own observation, and others have been reported by esteemed authors, that the anterior lobes of the brain may often be very gravely damaged without there being any sign of paralysis. It cannot be denied that his opinion is based on facts which cannot be contested. One need only read with care M. Broca's first case, that of *Tan*, to be convinced that the anterior lobes of the brain may be really damaged, without paralysis resulting. This patient, indeed, was completely aphasic for a period of ten years, without having manifested the slightest sign of paralysis of the limbs or of the face.

After that time his right limbs became paralysed, whilst after his death, dissection disclosed extensive lesions in the neighbourhood of the third frontal convolution as well as in the vicinity of the corpus striatum and the *insula of Reil*. Is it not evident, especially when this case is compared with the next one, that the Aphasia was due to the lesion of the frontal lobe, and the paralysis to that of the parts in the neighbourhood of the corpus striatum ?

The case which I am now going to relate, came under my observation in the first year of my medical career, and it made such a deep impression on me, that I have ever recollected it.

In the spring of the year 1825, two officers garrisoned at Tours fought a duel after a quarrel. One of them fired first, and the ball entered his adversary's head at one temple, passed through the brain, and then raised the temporal bone on the opposite side. Portions of the brain came out of the aperture of entrance of the ball, and some of them were found on the wounded man's hat. He was immediately brought to the Tours hospital, in a state of stupor; and although his breathing was easy, he gave no signs of consciousness. The left temporal muscle was divided, the piece of fractured bone raised with a spatula, and the ball extracted. As the operation was concluded, the patient made a sign with his hands, and expressed his thanks

in a very low voice. This terrible wound progressed very favourably, and after a few days the patient could speak and was not paralysed in the least. At the end of a month he could get about, and during the five months which he spent in the hospital, living almost constantly in the company of the resident pupils, he amused them by his merry ways and his witty conversation, whilst he employed his leisure by writing comedies. Towards the close of the summer, he complained of violent headache, and stupor supervened, with signs of acute cerebral softening, and on dissection, after death, a splinter of bone was found in the track of the ball, which had caused the inflammation of the cerebral tissue. The ball had passed through the two frontal lobes in their middle portion, and from the very first day after the infliction of the wound there had been no signs of paralysis, the patient could speak, and had never suffered from the least hesitation in the expression of his thoughts until the cerebral softening, which caused death, supervened. Mark, gentlemen, that this autopsy was made in 1825, at a time when nothing was said about the effect of an injury to a special portion of the frontal lobes. This remarkable case proves, therefore, on the one hand, what Dr. Auburtin maintains—namely, that grave lesions of the frontal lobe may be present without inducing paralysis; whilst, on the other hand, it shows that if lesions of the frontal lobe bring on Aphasia, the lesion must at all events involve a special portion, probably the one indicated by M. Broca. Professor Bouillaud's doctrine as to the frontal lobes being the seat of the manifestations of thought by speech, writing, and gesture, remains entire then, whilst M. Broca has the credit of having localised the faculty more exactly.

The following case, observed by M. Peter, at the military hospital of Gros-Caillou, may be placed by the side of the above. A drunken cavalry soldier fell from his horse, on the back of his head, and fractured his skull. Stupor set in at once, followed afterwards by the most violent agitation and delirium. The man kept constantly shouting the worst possible oaths, and held connected conversations with imaginary persons. He died at the end of thirty-six hours, without having recovered his reason. On dissection, a fracture of the roof and base of the skull was found in all its length. The most remarkable fact was, that, although the man had fallen on the back of his head, as was shown by the bruising of the soft parts and the starred fracture of the occiput, the brain was not injured at that part; whilst its anterior lobes were in a pulpy condition, through a most violent contusion evidently caused by the knocking of the cerebral mass against the anterior portion of the cranial vault. The whole thickness of the lobes was disorganized, and the alteration extended on each side as far as the anterior origin of the furrow

of the olfactory nerves. This case again showed that the two frontal lobes may be destroyed in their anterior portion, without causing a loss of the faculty of speech.

It is important to inquire whether the *intellect is damaged* in aphasic individuals, and to what extent it is so; but it is difficult to estimate this. A remarkable fact is, that such patients have usually an intelligent look, and, by a few gestures, supply the absence of spoken expressions which they cannot command. In order to estimate their intelligence, then, we can only use as our guides the expression of their face, writing, and gesture. The face, as I have already told you, does not differ much from its usual aspect, and so far, it would seem that the intellect is unimpaired; but to this I must oppose this remark. It must have often occurred to all of you to speak to a dog, and to question him as it were. You must, certainly, have been struck then with the bright look, the vivacity, and the singularly intelligent expression of the animal; with the manner in which he moves his head, and often also with the low cries, the emphatic grunts, with which he accompanies this pantomime. You must often have talked to him, and often exclaimed, "He only lacks speech." Well, gentlemen, apply this remark to an aphasic individual, and you will be convinced that there is less expression in his face than in that of a dog, and you will then admit that some other signs are required for judging of a man's intelligence.

Writing can help us; but most aphasic individuals are paralyzed on the right side, and cannot write. If they learn to write with the left hand, however, it can be easily seen that they cannot write a greater number of words than they are able to articulate. You have seen the many trials to which I subjected my patients. The young man Henri Guénier could sign his name with his left hand, when asked to do so; Paquet could do the same; but you may remember that Guénier's vocabulary consisted of two words, *yes* and *mamma*, and when I asked him to write down *yes*, or *mamma*, he always wrote his name. He had, with great pains, been taught to sign his name, and the motor muscles of his hand had got used to it, in a sort of automatic manner, and continued to act in the same way when he was asked something else. Paquet, also, signed his name well with his left hand, and when he was asked to write down *fork*, he still signed *Paquet*. I made him copy the word *ward*, which was printed on his card, and he did so; with some hesitation, it is true, but still he managed to do it. But when I took away the card, and then asked him to write down *ward* again, he wrote *Paquet*.

You will agree with me that such limited manifestations indicate great weakness of intellect. You may also remember the

patient whom Dr. Lancereaux brought us. This man boasted of possessing an unimpaired memory, of being still able to read, or, at least, of understanding perfectly all that he read, and to be in full possession of his intellect, speech alone failing him according to his account. I asked him to read a letter which began in these words: "My dear master," and he read without hesitation, "Sir," and then stopped short. He mumbled a few incoherent words, as if he endeavoured to decipher characters which had no meaning for him; then seeing by chance the word "miss," he read "madam." It was evident that he could not read. I asked him to write the word "sir," which he had just read by mistake in the letter, and he slowly wrote his own name. I next tried to make him read the preface of a *History of Saint Geneviève*; and instead of "preface," he at first said "fasts," and then was unable to decipher the first sentence: "Four centuries have elapsed since a humble shepherdess." He pronounced the word "centuries" well, said "three" instead of "four," and singularly enough, whilst he said "three," he held out four fingers, so as to help, by a gesture, his impotent speech. On my reading myself the sentence aloud, he listened with a certain degree of attention, and at the word "shepherdess," he exclaimed, with a fatuous smile: "Oh! shepherdess, know well what it is; love well shepherdess; draw well shepherdess," always leaving out the pronoun "I," which he could not articulate.

As he is a painter, a pupil of Coignet, and boasts of still drawing very well, I asked him to draw a shepherdess. After three or four minutes spent in efforts which brought big drops of perspiration on his face, he only managed to draw with a pencil unformed features, which had no resemblance to anything whatever. He succeeded, however, in drawing passably a man's head, but such as would have been drawn by a child of eight years of age, who had not learnt to draw. Here is an individual, then, who pretends that his intellect is unimpaired, who says that he can read, write, and draw well, and who cannot, in reality, decipher anything, can only sign his name, and sketch a grotesque human head. In other words, the truth is that his fingers obey an automatic impulse, to which thought remains a complete stranger.

This man's intelligence presents strange gaps; he knows, for instance, what is meant by the word "strength," and is absolutely ignorant of what is meant by "weakness," which is, however, the correlative of strength. When I asked him whether he felt weak, he did not understand me; but on putting to him the same question indirectly, by asking whether he felt less strong, he understood. As to the word "weakness," he was not only unable to articulate it, but he had completely forgotten both

the word and its meaning. His sentences were, besides, of the most primitive kind, in the following style :—"Me always worked,—much worked;"—"me always first,—first—first." He had forgotten a great many parts of speech. I shall revert to certain particulars of this case by-and-by, when I speak of the psychology of Aphasia.

Memory, which is so important a faculty of the understanding, is deeply injured, as may be easily ascertained. Most aphasic patients answer very well by signs, and I have repeatedly made the following experiment in your presence. I show them a spoon, and ask them what it is. They make no answer. Is it a knife? They make a sign of denial. Is it a fork? A sign of denial again. Do you remember the name of the object which I am showing you? Denial. Is it a spoon? A very earnest sign of approval. And so it is with nearly all aphasics. This is singular, however, that although they do not remember the name of the object, they perfectly remember the use of it. When shown a spoon, and asked what it is meant for, they take and raise it to their mouth, in order to indicate its use.

M. Lordat, who holds spiritualistic doctrines, and believes in the absolute independence of thought and speech, and *à fortiori*, in the independence of thought and the organs of speech, furnishes in himself the proof of their mutual dependence. Before he was attacked with Aphasia in 1828, he improvised his lectures admirably; but after his perfect recovery, he became incapable, not of improvising only, but even of reciting from memory, lectures which he had written beforehand, and he was obliged to read them.

There is no doubt, therefore, that the intellect is deeply injured in Aphasia; and when the affection gets well under our eyes, as it pretty frequently happens, we witness day after day the resurrection of the faculties, and we see them progress exactly as in the convalescence of a grave disease we see physical aptitudes return day after day.

But when the Aphasia is temporary, the testimony of the patients becomes very precious. Those very individuals whose intellect seems to be least impaired, have still lost some of it. Recall to mind the case of my colleague, who was aphasic for a few hours only, and who remembered so well the curious phases through which his mind had passed. He was seized while reading one of Lamartine's *Literary Conversations*. This is not fatiguing reading which requires great attention, and yet he noticed that *he did not understand* well what he was reading. He put his book down for a while, but on taking it up again, he made the same remark. On then trying to speak, he could not utter a single word, and on attempting to write, could not manage it either; yet, alarmed by these symptoms, he moved

his arms, his tongue, and thus ascertained that he was not paralyzed. He even collected his thoughts, and asked himself what portion of his brain could be, at that moment, injured. His intellect, therefore, was of a greater range than that of many men, and yet it was impaired, as proved by the difficulty which he had in understanding a page of Lamartine.

You recollect the woman Keller. She seemed to have recovered her intellect, answered with ease the simple questions which I put to her, and read part of the day. But when she was thoroughly well, I asked her to appreciate herself the state of her mind during her illness. She confessed that her memory was not so good, that she did not understand so well what she was told, that she had lost much, and that she *read with her eyes* well, but not *with her stomach*, a *naïve* and singular expression, by which she meant to designate her intellectual failure, whereas the organs of the senses acted to perfection.

Adèle Ancelin, also, read the whole day, and so did Paquet; so that those among you who are the champions of the intellectual aptitudes of aphasics, bring forward, as a powerful argument, the fact that the patients read attentively. Adèle Ancelin had, for a year, the same book in her hands, a religious book, the *Month of Mary*. The poor girl almost always read the same page, a fact which showed that she could not understand what she read. On several occasions, as you may remember, I took her book, and read aloud the very page which she had constantly under her eyes; and when I asked her if she understood what I read, she shrugged her shoulders to express that she did not. Paquet had received a pretty good education, since, as I have told you, he was going to be ordained when he left the seminary. He sometimes reads a whole day, and I must confess that he follows pretty well the lines in the book, that he turns the pages correctly, and seems to understand perfectly. But the following experiment proved categorically that he understood much less than he seemed to do. I took up his book, and read aloud a few lines at the bottom of a page, asking him to follow me with his eyes, and to turn the page when I came to the last line, but he never could do it correctly. Now, a child of five years of age, who can read, will turn a page properly, however limited his intelligence may be. Another circumstance shows, besides, that if Paquet understands perfectly what he reads, he has no recollection of it, and it will be easily admitted that memory is one of the most important faculties of the understanding, and that animals themselves possess it in an eminent degree. Paquet has collections of tales on the table by his bedside, which he reads over and over again, and always with the same degree of attention. Now, as a rule, one cares very little for a tale

which he has already read, and it would be an unbearable torture to be condemned to read the same tale thirty times a day. So that Paquet either does not understand what he reads, or does not recollect what he has just read, both hypotheses indicating a notable impairment of the intellect. He yet plays draughts and dominoes, and pretty well too; he is even guilty of cheating, an act which requires some degree of cunning; and when his adversary finds it out and compels him to begin again, he either grows impatient or laughs as if in joke. This very man, however, who plays these games, and makes pretty learned combinations, is incapable of counting his age on his fingers.

Insane individuals possess the same aptitudes. I have always been struck with the speciality of each man's intellectual aptitudes; but I have never understood the signs of extraordinary intelligence often exhibited by individuals who are completely demented.

When I was a resident pupil at Charenton, in 1825 and 1826, I often went to the drawing-room in the evening, and played some game with the insane patients. I have always been a bad player at draughts and chess, but I was vexed to be easily beaten by persons who could not put two ideas together. I was a better hand at backgammon, but I had not better luck when I played with individuals who had been formerly very good players. I was surprised at it, and even now, when I think of it, after an interval of nearly forty years, I cannot conceive how, in a brain so deeply disorganized as that of a demented person, combinations can be formed superior to those of a man of sound mind. I should not be astonished in the case of a monomaniac, because he is delirious on a pretty limited point only, so that he may retain all the aptitude which he may have previously possessed for certain games or for calculations; but I cannot understand how the very varied combinations of a game of cards or backgammon can be made in the mind of a maniac, who seems incapable of putting two ideas together.

To sum up, then, I maintain that aphasics are, as regards intelligence, considerably beneath the generality of men, and especially considerably beneath their former selves, when the comparison can be instituted. There is, however, a form of Aphasia in which the intellect is unaltered. Memory is good, the patient writes easily, and expresses his thoughts correctly in writing, as educated deaf-mutes do. This form is very rare, and it has seemed to me to differ so widely from the other, that I have thought myself warranted in regarding it as a distinct variety, particularly as in all the cases of the other form of the disease, the inability to write is proportionate to the inability to speak. The following case struck me the most.

I received one day, in my consulting-room, a carrier of the Paris

Halles, very young, and having the appearance of a man enjoying excellent health. He made signs that he could not speak, and handed to me a note, in which the history of his illness was detailed. He had written the note himself, with a very steady hand, and had worded it well. A few days previously he had suddenly lost his senses, and had been unconscious for nearly an hour. When he came round, he exhibited no symptom of paralysis, but could not articulate a single word. He moved his tongue perfectly—he swallowed with ease, but, however much he tried, he could not utter a word. Thinking that faradization might be of some use to him, I transferred him to my friend Dr. Duchenne (de Boulogne). He was ineffectually galvanized for a fortnight; but, without any special treatment, he completely recovered his speech five or six weeks after the invasion of the complaint. It is very remarkable, however, that during the whole course of this singular affection, he could manage all his affairs, continue them even in a certain measure, by substituting writing for speech.

The following interesting case is something like the above. A lady, residing at Boulogne-sur-Mer, was for ten or twelve years an object of curiosity in the town and the subject of every conversation. She had a very peevish temper, and the country people said that she was bewitched, on account of her ill nature. After an accident (the nature of which I have not been able to ascertain), she lost her speech, and only retained the faculty of uttering the oath, "Sacré nom de Dieu," by which she expressed all her thoughts, whether sad or gay. The curious point about her was, that for a great many years she was able to manage some rather important business, and keep her house in very good order. She went to market herself, made her own purchases, bargaining for them by signs, and occasionally exclaiming, "Sacré nom de Dieu." It never occurred to any one that she was insane, and her friends never tried to obtain her interdiction, although they may have perhaps desired it. She was not paralyzed. I do not know whether, like the carrier whose case I have just related to you, she could write, and thus express her thoughts in writing.

There is another form of Aphasia which is sometimes met with after acute diseases, and which is characterized by the complete forgetfulness of words. The following is a remarkable instance of this:—

Madame M——, who usually enjoyed excellent health, and was endowed with a very remarkable intelligence, was attacked at the age of fifty-six with erysipelas of the face and scalp. For a few days somewhat grave cerebral symptoms manifested themselves, and when the fever disappeared she had completely forgotten all words. For several days she remained in a sort of

automatic condition, accepting food and drink without asking for them, and giving expression to no thoughts. After a few days she could repeat, using them in their proper sense, words which were told her. Shortly afterwards she began to put a few words together, so as to construct parts of sentences or very short sentences: at that time she had completely recovered her physical health. At first she merely repeated the words which she was told, but after a time, her memory, by degrees, suggested others to her. She asked for paper, pen, and ink, and spent several hours every day, for a period of three months, writing down all the words which she could remember. I have seen her manuscripts, and it was curious to observe by what process one word reminded her of another; sometimes the first, sometimes the second, syllable gave her the key to the next word. The rhyme often suggested words to her, and sometimes a very distant meaning. The following examples will illustrate this:—"Cat, hat; hand, sleeve; gown, petticoat; rose, nose, nosegay; beer, froth; rope, well, ditch," &c. &c. She thus covered nearly five hundred pages with small text.

My colleague, Dr. Boucher, Professor of Medicine in the Dijon Preparatory School of Medicine, has since observed two cases of this kind, in the course of an epidemic of typhoid fever which desolated the town in 1863.

The son of the porter of the Imperial Lyceum at Dijon, aged thirteen, of a delicate constitution, was seized with fever in the month of September. His life was in danger for some time, but the symptoms became more favourable at last, and all was going on well, when complete Aphasia supervened one morning. It was a sad, though curious sight, to see the supreme efforts of the child to articulate the simple word "no." Dr. Boucher tested the urine for albumen, and detected its presence in a small quantity. As the general symptoms, however, continued to be good, tonics and proper nourishment were insisted on. After four or five days the words returned by degrees, although enunciated with remarkable slowness; but at length complete recovery took place, and the boy, after a somewhat lengthened convalescence, resumed his studies at the school.

The second case observed by Dr. Boucher was that of a child aged three years, who had presented very grave nervous symptoms during an attack of typhoid fever, and who likewise passed albuminous urine. Speech was also lost suddenly, when the fever ceased to be dangerous: convalescence was very much prolonged.

You probably remember the case of a woman who was in my wards in the year 1863, and in whom we observed, after she had had a serious attack of typhoid fever, symptoms exactly like those described by Dr. Boucher.

It is not a rare thing to meet with paralyties who cannot distinctly articulate, who sputter out their words, and whose tongue is so embarrassed as to render them incapable of expressing the few thoughts which they have. With a little attention, however, it is easy to see that a particular intonation corresponds to each thought, so that the persons about such patients understand pretty well, after a time, these imperfect grunts. The patients use correct words for answering questions, but the paralysis of the organs of speech prevents them from articulating distinctly. The same thing occurs in glosso-laryngeal paralysis. In ordinary paralytics the intellect is deeply damaged, but in glosso-laryngeal paralysis the intellect is unaltered, the patient can read and write, and it can be easily seen that, when they try to speak, their eyes and gestures make up for the incompleteness of their speech. They have, therefore, at the command of their intellect, all the manifestations which a healthy man can dispose of, save speech, the impediment in which is proportionate to the degree of paralysis of the organs which are used for articulate language.

I can well imagine that all these distinctions appear somewhat subtle to persons who read books without seeing patients; but when they are studied at the bedside, they are so strikingly manifest, that even those among you who have just begun their medical studies catch at once the shades which separate affections which at first sight appear to be identical. There is another curious character by which the ordinary paralysis of Aphasia may be distinguished from glosso-laryngeal paralysis, and the loss of speech which is a consequence of the total loss of memory; namely, the excessive emotional sensibility which is usually found in true paralyties. A person who has become hemiplegic after an attack of cerebral hæmorrhage is constantly shedding tears, for the least thing; and this is a symptom which has been noted by all observers. But in Aphasia this symptom is in most cases absent,[1]

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[1] In some cases of Aphasia with paralysis, when the degree of hemiplegia indicates extensive disease of the brain, the emotional excitability of the patient may be as considerably marked as in ordinary cases of hemiplegia. This condition manifests itself not only by a greater proneness to shedding tears than the occasion warrants, but by a greater tendency also to immoderate laughter, and to a rapid, sometimes sudden and unexpected, transition from laughter to tears. In Aphasia, however, notwithstanding these symptoms of cerebral disturbance, the patient's face will retain its intelligent aspect, and will not have that heavy, dull look of individuals afflicted with chronic paralysis of cerebral origin. These remarks are suggested by the following case, which came under my observation at the National Hospital for the Paralysed and Epileptic. The patient was at first admitted under the care of my colleague, Dr. Ramskill, who kindly transferred her to me.

M—— W——, aged twenty-four, a fair-haired, blue-eyed young woman, of short stature and pale complexion, but apparently well nourished, was admitted

and I confess that I cannot conceive the reason why. This proneness to shed tears is commonly ascribed to the grave impairment of the intellect, which usually accompanies paralysis; but

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an out-patient, at the National Hospital for the Paralysed and Epileptic, on the 10th of January, 1865, suffering from imperfect right hemiplegia and complete Aphasia. To all my questions, she invariably answered: "Sapon, sapon." She was accompanied by her sister-in-law, from whom I gathered the following particulars:—

The patient had been seized with paralysis on the right side three months previously. She had led an irregular, immoral life, and had committed various excesses (it could not be ascertained whether she had had syphilis). For a month previous to her seizure, she had complained of severe headache. The attack had been sudden: she dropped down senseless, remained in a comatose condition for several (?) days, and when she recovered her senses, could not utter a single word beyond "*Sapon, sapon,*" which she has, ever since, kept repeating at every turn. The paralysis was not complete after the first few days, and the face is said to have been involved.

When I first saw her, the patient had walked to the hospital, a distance of about two miles from her residence. She dragged the right leg much in walking, and was unable to stand on it alone. She could move her right arm, but not so as to use it, and she could not squeeze my hand at all. There was no rigidity, no wasting of the limbs. The facial paralysis had disappeared, for the patient could show her upper teeth, when asked to do so, and there was no deviation of the angles of the mouth when she laughed. There was no cardiac disease; the heart's impulse was natural, and its sounds normal, both at base and apex. The patient's face was full of expression, and her eyes beaming with intelligence; yet it was manifest that these appearances were deceptive, and that her intellect was very much impaired. She could not be made to understand at once, by words alone, what was required of her; and could not always answer correctly by gestures the questions which she was asked. Her pantomime was not so clear as that of a deaf-and-dumb individual, and she seemed not to be able to understand the meaning of words. They had to be spoken very slowly, and repeated several times, before she could catch their meaning, and she most frequently failed completely in this. Gestures she understood at once. Thus, when I asked her to show me her tongue, she did not always do so immediately; but on my putting out my own tongue, and then making signs to her to do the same, she instantly complied. She protruded her tongue well, and although its apex pointed to the right, it could still be moved, at will, in all directions; thus showing that the loss of speech could not be ascribed to paralysis of that organ. Besides, the perfectly distinct manner in which she articulated the words *sapon, sapon*, proved that the defect under which she laboured was not one of articulation, but clearly of the faculty of speech. The fact that she did not understand what she was told in words, whilst she understood the meaning of gestures, showed also that the defect did not so much arise from want of comprehension, as from forgetfulness of words as the representatives or symbols of ideas. A peculiar circumstance in her case (which I have not met with in other cases of Aphasia that have come under my notice), was, that she was prone to shed tears, or to laugh immoderately for the least thing, as ordinary hemiplegics are well known to do, at a certain period of their complaint.

She could not write a single word with her left hand; she held her pen properly, but only made a meaningless scrawl, like those series of angular lines which a child who has never learnt to write makes on paper, when he plays at writing. Although she kept constantly repeating *sapon, sapon*, I could never make her say *sap* or *pon* by itself, or repeat any syllable or word after me. She knew her own name, and when I mentioned it she laughed, and pointed to her-

in cases of Aphasia, there is as deep an impairment of the intellect, and yet there is more rarely found this tendency to whining. I lately saw, at the Maison Municipale de Santé, with my colleague Dr. Bourdon, a merchant suffering from complete Aphasia, and who was easily moved to tears. There were two things in his case, however; for he had had two successive attacks of paralysis, one on the left, and the other on the right side. He had become aphasic after the second attack, and since the first seizure, which had presented the same symptoms as the generality of cases of cerebral hæmorrhage, he had been prone to shed tears on all occasions, and this had not disappeared since he had become aphasic.

In Aphasia, the inability to speak depends on very various causes, which it is very difficult to analyze well. There is, in the first place, *amnesia*, as can be seen at a glance, and, in most cases, this is even the most prominent symptom. The patient does not speak, because he does not remember the words which express

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self. According to her sister's statement, she remembered localities, and knew faces well.

A month after she first came under my observation, she suddenly grew worse, and exhibited symptoms of acute cerebral inflammation. She became feverish, pointed to her head, as if in pain there, and grew at first very excitable and then delirious, whilst rigidity of the right arm and leg set in, and, at one time, nearly incessant spasmodic agitation of those limbs. Under the influence of mercury and iodide of potassium, these acute symptoms gradually gave way, but left behind them a greater degree of paralysis and rigidity of the limbs. The intellect seemed also to have become more clouded than before. As to the faculty of speech, no modification was noticed; she still kept repeating *sapon, sapon*. By slow degrees, however, and after the lapse of many months, she improved remarkably, and at the present date, October, 1865, she is considerably better in many respects. She scarcely drags her right leg when walking, and can squeeze pretty hard with her right hand. There is still some slight rigidity about the arm, and she cannot use it sufficiently well to admit of her dressing without the help of another person. Her intellect is improved, but not in the same proportion as the paralysis. Her emotional excitability is much less than before, although it is still marked. Her vocabulary comprises now a few more words. She still says *sapon, sapon*, but she can now distinctly articulate *yes* and *no*, although she does not always use them appropriately, and she can count *one, two, three, four*. When under the influence of great excitement, she sometimes exclaims, "Oh, dear me!" according to her sister's account. She cannot yet write a single word, nor even form a single letter, although she has often tried hard. She does not know the letters of the alphabet, and when she is shown *a* and *o*, and asked to point out *a*, she cannot do it. She has still great difficulty in understanding what is said to her in words, although she is not in the least hard of hearing; but she immediately understands gestures. Her own pantomime still lacks clearness. She never reads, but is fond of looking at pictures. A short time ago, however, she looked over a batch of old letters, seemed to read them, and threw some into the fire, and kept others. On that day her mind may have been clearer than at other times, for the persons about her have noticed great fluctuations in her intellect, and have sometimes been led, through this, to hope that she would recover. Of late, she has complained of failure of sight of the right eye; but her general health appears to be excellent, and she has gained flesh.—Ed.]

ideas. You recollect the experiment which I often repeated at Marcou's bedside. I placed his nightcap on his bed, and asked him what it was. But, after looking at it attentively, he could not say; and exclaimed, "And yet I know well what it is, but I cannot recollect." When told that it was a nightcap, he replied, "Oh! yes, it is a nightcap." The same scene was repeated, when various other objects were shown to him. Some things, however, he named well, such as his pipe. He was, as you know, a navvy; and, therefore, worked chiefly with the shovel and the pick-axe, so that these are objects the names of which a navvy should not forget. But Marcou could never tell us what tools he worked with, and after he had been vainly trying to remember, when I told him it was with the shovel and the pick-axe, "Oh! yes, it is," he would reply, and two minutes afterwards he was again as incapable of naming them as before. There exists, therefore, a loss of memory sufficiently great to prevent the patient from being able, of his own accord, to designate an object by its proper name, but not sufficient to prevent his remembering the name when uttered in his presence. Thus, this same patient, Marcou, who could never spontaneously tell us the name of his nightcap, recognized its name perfectly, however, when mentioned in his presence. When it was shown him, and was called a pipe, or trousers, he exclaimed, "Oh! no," whilst he replied "Yes" when asked whether it was his nightcap. The condition of such patients is, therefore, analogous to that of a schoolboy whose memory fails him when reciting a lesson. If the master prompt him, he begins again, and continues without a mistake, provided that he is endowed with a certain degree of memory; but if he has no memory at all, every word has to be told him. On this condition alone, can he recognize and repeat the words which he has learnt, or tried to learn. In some cases, memory is so impaired that the patient does not feel positive as to the name of an object which is shown to him. Paquet, as you know, was very deficient in this respect. When told that he was wrong, after he had nodded approvingly on an object shown to him being called by its correct name, he hesitated, and looked perplexed. Marcou, however, was never shaken in his confidence.

It is undeniable, therefore, that there is a loss of memory, and this symptom sometimes constitutes the only morbid phenomenon. In the month of January, 1864, one of my colleagues in the Academy of Medicine was seized, on returning home, with slight vertigo. He tried to speak, and found that he could not remember a great many nouns! Drs. Pidoux and Const. Paul, who were sent for, easily recognized this singular disturbance of the intellect. The patient suddenly stopped in the middle of a sentence, unable to express a noun; he hesitated, manifested

impatience, and if the word he wanted was pronounced by another, he exclaimed "That's it," and repeating the word very distinctly, he continued his sentence. In this case, then, most of the parts of speech were perfectly remembered, but nouns were completely forgotten. Let us note, however, for it is an important fact, that the patient caught the word at once, as soon as it was pronounced, and articulated it with extreme facility.

But there is, in Aphasia, another very strange phenomenon, which is, perhaps, another form only of amnesia; it is the inability to articulate words, however hard the attempt. Paquet, for instance, can say *cousisi*, and it seems, therefore, that he might easily say *cou-cou*, or *sisi*. But you saw that for several days in succession, I stopped a good while by his bedside, and that it was only after a few days that I could succeed in making him say *cou-cou;* whilst he never could say *sisi* alone. The same remark has already been made by Dr. Perroud, physician to the Hôtel-Dieu at Lyons, who had under his care an aphasic woman who could say very well, "Bonjour, monsieur," but could never be made to say *bonbon*, a word which is, after all, the repetition merely of the first syllable in *bonjour*. You saw, also, what pains I took to make Paquet repeat a few syllables. He could say *a* pretty easily, but could never say *pa*. I told him to imitate the movements of my lips as I pronounced the letter *p*, but he could not succeed in doing so. I tried again to make him say at first *peu a*, in hopes, after a time, to make him by elision contract the two sounds into the single one *pa;* but I failed completely.

It would seem, therefore, that there is, in these cases, an inability to co-ordinate the movements which are needed in phonation; for the patient has fully retained the power of moving his tongue and lips in every direction, as may be easily ascertained, although he is incapable of making combined movements in order to enunciate a word. I have asked myself whether there was not merely forgetfulness of the instinctive and harmonious combination of movements which we all learn in infancy, and which constitutes articulate language; and whether an aphasic patient was not, consequently, in the same condition as a child who is taught to speak for the first time, or of a deaf-mute, who, on being suddenly cured of his deafness, tries to imitate the speech of the persons whom he hears for the first time. There would then be this difference between an aphasic and a deaf-mute, that the one has forgotten what he has learnt, whilst the other does not yet know. I am the more inclined to believe that this opinion is well founded, because there is, in nearly all cases, loss of memory of writing as well as of speaking. In general an aphasic individual is not more apt to express his thoughts in writing than in speaking; and although he has retained the power of moving his hands as nimbly as before, he is yet as unable to compose a word with

his pen as he is to articulate it. Now, in such a case, it is impossible to admit a defect of co-ordination, whereas loss of memory explains everything.

Another mode of expression of one's thoughts, namely, by gestures, is as deeply modified, in many cases of Aphasia, as speech itself. Now, when an individual moves about with the greatest ease, when his face is variously agitated according as he is under the influence of joy, surprise, or pain, it is strange that his face should be incapable of feigning the expression of these same feelings when they are not really felt. Thus, an aphasic cannot, when asked to do so, put on the face of a person who is crying, and his inability cannot be due to a defect of muscular co-ordination, since, when he feels real grief, the expression of his face clearly shows it. You saw me make the following experiment with Paquet: I held out my two arms and hands, and moved my fingers, like a man who is playing the clarinet, and I asked him to imitate me. He immediately executed the same movements with perfect precision. When asked whether he knew that the attitude was that of a man who played the clarinet, he replied affirmatively, by nodding his head. A few minutes afterwards, however, when asked to put himself in the attitude of a man who is playing the clarinet, he seemed to think, and, in most cases, was unable to reproduce this easy pantomime. There was amnesia, then, in his case, for he did not recollect.

The illustrious Professor Lordat, who has himself suffered from Aphasia, gave, after his recovery, an account of the inward sensations which he felt during his illness, which perfectly indicates the part played by memory. He could think, he could co-ordinate a lecture, or change its arrangement in his own mind, but he was unable, although he was not paralyzed, to express his thoughts in speaking or writing. "I thought," says he, "of the Christian doxology: 'Glory be to the Father, the Son, and the Holy Ghost,' and I was not able to *recollect a single word of it.*" Thoughts seemed to arise freely, but the mode of expressing them in sounds, the receptacle of these thoughts, was forgotten. In the experiment which I related to you just now, I evidently awakened Paquet's recollections, but he could not express them by gestures. I am anxious to give prominence to the fact, that in Aphasia loss of memory plays the principal part; the patient forgets, wholly or partially, the various modes of expressing thoughts, and is in the condition of a deaf-mute, who, on suddenly gaining the faculty of hearing, does not yet know how to use the organs of phonation.

But does it follow that *Aphasia* and *Amnesia* are, in my opinion, synonymous? Certainly not. The aphasic patient, who has forgotten how to express his thoughts in speaking, in writing, and by gestures, often remains apt to form the difficult combinations

needed in the games which tax memory much. They remember perfectly circumstances which occurred long ago; and you have heard how Professor Lordat, and that colleague of mine whose case I related to you, recapitulated, mentally, series of very complex ideas, although, according to all appearances, their intellect was perhaps not so clear as it had previously and as it has since been. Yet the fact of broad conceptions cannot be contested, of conceptions of a higher order, certainly, than those of uneducated and ignorant men, who, nevertheless, express themselves with readiness. It is assuredly very strange, that men who are evidently endued with a pretty extensive memory on certain subjects, should be completely devoid of all memory when they have to express their thoughts in speaking and writing, and by gestures.

You remember the stratagem practised by the hospital *employés* for finding out Marcou's name and address; you remember how he walked back to the stone-yard where he usually worked, so that he must have recollected localities. Again, you have seen Paquet play dominoes and draughts, remembering pretty difficult combinations, although, for the last eight months he has only been able to say *cousisi*. Dr. Lasègue knew a musician, who was completely aphasic, and who could neither read nor write, and yet could note down a musical phrase sung in his presence. Whatever be the share, therefore, which I am disposed to ascribe to loss of memory in Aphasia, I am compelled to admit that certain special kinds of memory are untouched. This statement may appear strange, for it might seem that there is only one kind of memory. But it is not so.

I knew a medical student, a good musician, but of ordinary intelligence in other respects, and not endowed with a retentive memory, who could, on his return home from the Opera, play on the violin all the airs of an opera which he had heard for the first time. You may have heard of Mondheux, the young shepherd in Touraine, who had such a wonderful memory for figures and calculations, that, if he were unexpectedly asked how many hours had lived a man aged forty-five years, four months, and five days, he could, in less than two minutes, give the correct answer, without having recourse to a pencil or a pen. His memory was, in other respects, of very ordinary range, and when he was a little older he evinced no aptitude for mathematics. One man has a good memory for localities, another for names, a third for dates or for figures, and each of these *varieties* of memory, if I may be allowed the expression, is independent of the rest. So that, if it were admitted that an aphasic patient suffers, after all, from loss of memory, it should be added that he has lost all recollection of the mode of expressing thoughts in writing, in speaking, and by gestures.

Now, I do not know how to classify those cases of Aphasia in which the patient is able to express himself in writing, though not in speaking, as in the case of the carrier which I have related to you. We must, in such instances, suppose that the patient no longer remembers the movements which are needed for producing and modifying vocal sounds. Thus, the man whom I saw with Dr. Duchenne, and who could give in writing proofs that his intelligence was still of a good order, could not even articulate the syllable *ba*. He moved his tongue and lips perfectly, he swallowed as well as before, and yet, when we stood before him, and asked him to say *bon*, to close his lips as we did, and then to utter the sound *a*, as we opened our lips, he made the most curious grimaces, but could not succeed in saying *ba*. Now, this very man, when eating or drinking, approximated or separated his lips with perfect regularity. There was, consequently neither loss of power nor disorder; this occurred only when he attempted to execute a determinate movement, as in the act of speaking; just as there are loss of power and disorder in cases of what Dr. Duchenne has called *functional spasm*.

Such patients, indeed, use their right hand very well to shave themselves, to play the piano, to sew, to pick up the most minute objects, but as soon as they try to write, the muscles of their hand instantly become affected with spasm, and their pen can only trace illegible characters. Again, a violin-player may write perfectly, but he either cannot hold his bow, or the hand which holds the violin is the seat of spasmodic contractions. Is this the case in this particular form of Aphasia? It may be objected that there is no spasm of the vocal organs in Aphasia, but loss of the aptitude in virtue of which the numerous organs concerned in the production of voice (the lips, tongue, soft palate, glottis, and the various parts of the larynx) act in harmony, in order to produce determinate sounds. It may be objected that there is loss of this complex co-ordination, which seems to us natural and easy only because we have forgotten the time and trouble which it has cost us; in other words, that there is what Dr. Lordat so justly terms *verbal inco-ordination*. There is not inco-ordination only, but *verbal amnesia* also; for the patient has lost the memory of words. Yet, has he forgotten words alone, apart from the ideas which they express? This question involves one of the most intricate problems in metaphysics; namely, whether ideas can exist independently of the words which represent them. I will not presume to settle definitively this problem, which has been solved in two opposite ways by spiritualists and sensualists, but will merely confess that I incline to the opinion advocated by Condillac and Warburton, that words are necessary, nay, indispensable instruments of thought.

I cannot, therefore, endorse Professor Lordat's view, that

thought is absolutely independent of speech, and that a subject may be limited, may be developed and divided into elementary ideas, which, in their turn, may be subdivided into simpler ones, although all recollection has been lost of the sounds which are used as signs, that is to say, when all memory is lost of the words composing language.

In support of his theory, Dr. Lordat quotes his own case. Although seized with Aphasia, he could, says he, combine abstract ideas, distinguish them accurately, *although he could not command a single word to express them, and without in the least thinking of this mode of expression.* "He was conscious of no impediment in the act of thinking. Accustomed to teach for a great many years, he congratulated himself on being able to dispose in his mind the chief points of a lecture, and on having no difficulty in introducing any changes which he liked, in the arrangement of his ideas."

But the celebrated professor will allow me to ask him whether he did not deceive himself? and whether he was not in the same condition as that patient of Dr. Lancereaux's whom I mentioned a short time ago, and who also believed that he was in full possession of his intellect? Yet, on putting it to the test, his intellect was clearly shown to be impaired; thus proving that he had not merely lost the physical faculty of the material transmission of his thoughts.

Doubtless the mind may wander a little at random, without being obliged to give a corporal form to its ideas; but as soon as it tries to make them concrete (a condition which I regard as *indispensable for their co-ordination*), it seems impossible to me, at least in my own case, not to clothe them in their material dress,—namely, words.

It will, doubtless, be objected that a deaf-mute evidently thinks, before he is taught the gestures, by means of which he will henceforth hold communion with his fellow-men and improve his mind. But is it clearly proved that an untaught deaf-mute is apt to form conceptions of a very high order? Is it not probable that he makes use, even for the *elementary* ideas of which he is capable, of the material images of things instead of words, the images of ideas, as we do? Is it not probable that, when he thinks of a tree, for instance, he pictures to himself a tree by the image of the tree itself, instead of thinking, as we do, of the word *tree?* See, then, how inferior is the memory of an untaught deaf-mute, when compared with ours. Like him, we can remember a tree by the image of a tree, but we are also reminded of it by the word *tree*, which pictures itself to our mind, because we have read and written it. One may imagine how fettered thought must be in such cases, and how rudimentary intellectual conceptions must therefore be. A great thinker, as

well as a great mathematician, cannot devote himself to transcendental speculations, unless he uses formulæ, and a thousand material accessories which aid his mind, relieve his memory, and impart greater strength to thought, by giving it greater precision. Now an aphasic individual suffers from verbal amnesia, so that he has lost the formulæ of thought.

I believe that the same thing obtains in metaphysics as in geometry. In the latter case a man may vaguely conceive space and infinity without any precision or measure, but if he wishes to think of the properties of space, and more particularly of the special properties of the figures which bound space, as, say, conic sections, it is impossible that his mind does not immediately see the curves proper to a parabola, an hyperbola, and an ellipse. In metaphysics, on the other hand, I believe that a man cannot think of the special properties of beauty, justice, and truth, for instance, without immediately giving a material form, as it were, to his thoughts, by using concrete examples, and without associating words together—words which represent concrete ideas, and which then stand in the same relation to particular metaphysical ideas as figures do to determinate geometric ideas.

In Aphasia, therefore, there is not merely loss of speech, but there is also impairment of the understanding. The patient has lost simultaneously, in a greater or less degree, the *memory of words, the memory of the acts by means of which words are articulated,* and *intelligence.* But he has not lost all these faculties in an equal degree, for the understanding is less injured than the memory of the acts for producing sounds, and this latter faculty less impaired than that of remembering words.

To sum up, Aphasia consists in loss of the faculty of expressing one's thoughts by speech, and in most cases, also, by writing and by gestures.

As every distinct faculty presupposes a special organ, the advocates of localization made out that the seat of this faculty in the brain is the posterior portion of the third frontal convolution, chiefly on the left side. But the most varied lesions of this spot, and I will add of neighbouring parts, more deeply situated, such as the insula of Reil and the corpus striatum, can bring on Aphasia. Hence the same *prognosis* does not apply to Aphasia occurring during convalescence from a grave fever, and to Aphasia due to softening of, or hæmorrhage into, the brain. In the former case we can scarcely conceive the nature of the lesion, and it must be confessed that this form, which is essentially transitory, differs greatly as to its progress and termination from the persistent affection of which I have given you illustrations. In those cases where the affection is transitory, or is more or less prolonged, but is *unaccompanied by hemiplegia,* there may perhaps have been mere congestion. But however this may be,

these two forms must be carefully distinguished from a third, in which there is concomitant hemiplegia, for this last is, in most cases, absolutely incurable, or at best admits of very slight amelioration only. Another fact which should be made prominent, is the frequent termination of Aphasia in rapidly fatal apoplexy. This occurred in the instance of the woman Desteben, in that of the gentleman whom I went to see in the department of Landes, and in that of another patient whose case was mentioned to me.

It is undeniable that blood-letting has been immediately followed by happy results, but only in cases of Aphasia without hemiplegia, that is to say, when the lesion was not probably very deep. In cases of Aphasia with hemiplegia, unless dependent on syphilis, as in Marie K——, I must confess that we are almost completely powerless. We can no more cure the Aphasia than we can the paralysis which accompanies it. Nature alone, or nearly alone, brings on improvement, which is in all cases merely partial. The intellect of an aphasic patient is for ever damaged, just as the motility of one half of his body is impaired, and he will always be lame mentally.

[Note.—See a very interesting paper by my colleague, Dr. J. Hughlings Jackson, "On Loss of Speech: its association with Valvular Disease of the Heart, and with Hemiplegia on the right side," etc. etc., in the London Hospital Reports, vol. i., 1864, p. 388.

The subject of Aphasia has very lately (May, 1865) given rise to a prolonged and interesting discussion at the Academy of Medicine, of Paris. Like most discussions, however, it has succeeded in convincing neither party, and has left matters almost on the same footing as they stood before. If anything, the advocates of the localization of the faculty of speech in the anterior lobes of the brain have come a little worse off than their adversaries. Professor Trousseau, in a most able and brilliant address, summed up the cases and arguments given in the above Lecture, and maintained the views therein advocated; whilst Dr. Bouillaud obstinately refused to admit the authenticity of the cases which were brought forward in refutation of his doctrine. In the course of this memorable discussion, however, two new cases were mentioned, one of which decides entirely against Dr. Bouillaud's views, and the other against the doctrines propounded by Dr. Marc Dax and Dr. George Dax, and the more recent one advocated by M. Broca. The first of these cases was observed by Professor Velpeau, many years ago, and was at the time communicated by him to the Academy of Medicine, and subsequently published in the *Bulletins* of that Society. M. Longet has given a summary of the case in his work, "On the Anatomy and Physiology of the Nervous System," vol. i. p. 683.

The patient, a hair-dresser, was admitted into the Charité Hospital, suffering from incontinence of urine. He was an extremely tiresome talker, and died three weeks after admission, without having presented any symptom of cerebral disease, any difficulty of articulation, or defect of speech. On dissection, there

was found hypertrophy of the prostate, stricture of the urethra, and old disease of the mucous membrane of the bladder. The head was examined, merely in order that the autopsy should be thoroughly complete. The dura mater was found to be firmly adherent to the mass of the brain. The *right anterior lobe of the brain was completely destroyed* by a voluminous tumour, having all the characters of scirrhus. The *left frontal lobe*, also, had been encroached upon, and was, to a great extent, destroyed.

The next case was observed by M. Aug. Bérard, and communicated by him to the Anatomical Society of Paris, in 1843. The patient—a miner—was injured by the explosion of a mine. He was knocked down, and got covered over with *débris*. He did not lose consciousness, managed to creep out of his hole, and called to his help some men who were working at a short distance further off. He begged them to fetch a cart, and to take him on to M. Bérard's house, at St. Maurice. He was there examined by M. Bérard. The whole frontal region was laid open, the integuments hung in shreds, the bones were splintered and in detached fragments, and the brain was exposed. *Both anterior cerebral lobes were completely destroyed*, and in their stead was a mixture of blood, of bony splinters, and of brain substance. In spite of this frightful injury, the man could *relate in all its details how the accident had occurred.* From St. Maurice he was carried to the St. Antoine Hospital, where he died on the next day.

The following passage of Dr. Watson's classical work, "On the Principles and Practice of Physic," vol. i. p. 520, fourth edition, is, therefore, just as applicable now as it was a few years ago :—

"Gall had conjectured that the faculty of speech was placed under the governance of the anterior lobe of the brain ; and Bouillaud has endeavoured to support that opinion by a number of facts observed in connexion with cerebral hæmorrhage ; but Cruveilhier has brought forward several curious instances in which the loss of speech was a prominent symptom, while the disease was not found in the anterior lobe, but in some other part of the brain."

"Andral, with his accustomed industry, has accumulated evidence upon this point also. In thirty-seven cases of cerebral hæmorrhage observed by himself or by others, in which the morbid condition occupied one or both of the anterior lobes, the power of speech was affected twenty-one times, and unaffected sixteen times. On the other hand he has collected fourteen cases, in which the power of speech was lost, yet no alteration had taken place in the anterior lobes. In seven of these fourteen cases the lesion was situated in the middle lobes, and in the other seven in the posterior lobes of the brain. . . . . Since the loss of speech is occasionally the only, or the most prominent symptom, while in other cases the speech is not affected at all, we cannot but believe that this faculty is under the special guidance of some definite part within the cranium. But the facts that I have just been quoting show in the most convincing manner that we are not able as yet to allot these separate functions to their proper spots in the cerebral mass."

I extract the following from a remarkable lecture "On the Importance of the Application of Physiology to the Practice of Medicine and Surgery," delivered by Dr. Brown-Séquard, before the College of Physicians of Ireland, on February 3rd, 1865, and published in the *Dublin Quarterly Journal of Medical Science*, May, 1865 :—

"There is another kind of hemiplegia as to which I must say a few words—I

mean that which is due to a lesion of the anterior lobes of the brain. Phrenologists, you know, have regarded the anterior lobes as the organs of speech ; but there have been many instances—Dr. Stokes mentioned a very remarkable one to me a few days ago—in which there has been destruction of these parts without any deprivation of speech. But the question remains (and it is an interesting one), what occasions the loss of speech, when such loss takes place ? As regards this question I shall, in a moment or two, have to point out how great a variety of symptoms may be produced by a lesion of almost every part of the brain. The deprivation of speech I hold to be a reflex phenomenon ; and that it is so, we have almost a proof in the fact that it often varies very much in the same patient, according to circumstances which physiology has, as yet, been unable to detect, but certainly with a lesion of the brain still continuing unaltered. It is worthy of remark, too, that the loss of speech is usually unaccompanied by any loss of movement in the tongue ; there may be perfect freedom of motion in the tongue, and the deprivation of speech arises from the circumstance of the patient being *unable to give expression to his thoughts ;* and this inability extends not merely to speech : he is equally powerless to express ideas either by signs or by writing. The paralysis, in fact, is a 'paralysis of the organ of expression of ideas ;' and it is remarkable that this occurs, while the individual may remain, in other respects, in full possession of his intellectual faculties, at least so far as we can judge of this possession. One of the cases of that kind I have seen, was that of a clergyman, a man of remarkable intelligence. He had not lost the mechanical part of the power of speech, for he articulated a few words very distinctly, but they were sounds devoid of meaning ; he was equally unable to express his thoughts by writing, or even by signs. Even when he was told to express 'yes' by lifting up one finger, and 'no' by lifting up two fingers, he was unable to do it, and showed signs of great distress at his inability ; and all this, although he appeared, in other respects, very intelligent."—Ed.]

LECTURE VIII.

PROGRESSIVE MUSCULAR ATROPHY.

Pathological Anatomy.—Lesions of the Muscles.—Lesion of the Nervous System.—Is the latter constant?—Symptoms.—The Atrophy generally begins in the Upper Extremities.—Of the exceptions to this rule.—Prognosis, fatal.

GENTLEMEN,—I am enabled to-day to complete the details of the autopsy, which we made a few days ago, of a patient who died at No. 10 in St. Agnes Ward. M. Ch. Robin, who kindly undertook to make a microscopical examination of the muscles, has sent me a report which I am going to read to you. I wish first to state, however, that the autopsy was made in my presence by M. Sappey, who is at the head of the anatomical department of our Faculty, and that this skilful anatomist found no other lesion, besides the alterations of structure of the muscles, than marked atrophy of the anterior roots of the spinal cord, and *perhaps* a slight diminution in size of the anterior columns.

M. Robin's report is as follows:—

"In all the muscles, even in the palest, there is found a certain number of fasciculi, the fibres of which are marked by transverse striæ, although these striæ are, it is true, paler and less distinct than they normally are. The fasciculi of the diseased muscles are one-third less in diameter than the bundles which have retained their normal structure, and which have a diameter varying from $\frac{7}{100}$ to $\frac{12}{100}$ of a millimetre. The granular condition, paleness, and transparency of the atrophied bundles are most marked in the spots where their diameter is most notably diminished. This fact is particularly striking in the interossei muscles, in which a pretty large number of bundles are seen, the sarcolemma of which is completely empty, and so collapsed as to be reduced to a diameter of from $\frac{8}{1000}$ to $\frac{10}{1000}$ of a millimetre. The paleness and transparency of these muscles is remarkable. By the side of fasciculi which are not very atrophied, some are seen in a perfectly granular condition, and others still marked by very pale striæ; lastly, the most atrophied bundles are mixed up with others presenting, nearly all of them, the same degree of atrophy. There is no lesion of the intermuscular cellular tissue. *Fat-cells are not seen in greater numbers than normally.* The degree of paleness of the muscles is evidently proportionate to

the number of bundles presenting varying degrees of atrophy, from a simple diminution in size, with indistinctness of the transverse striæ, up to a granular condition without apparent striæ, and with extreme diminution of size.

"Fat-cells are found in as large numbers in the reddest muscles (which yet present a few bundles that are smaller and paler than the rest, and some of which are already granular and have lost their striæ) as in the palest ones, such as the interossei muscles of which I have spoken, and the muscles of the hypothenar eminence. This anatomical fact was most apparent in the intercostal muscles, a very small proportion of the fasciculi of which have become pale, and in which the granular condition is still more rare."

This case, gentlemen, so far as the results of a microscopical examination of the diseased parts are concerned, cannot, according to M. Duchenne (de Boulogne) be regarded as a perfect type of progressive muscular atrophy, or the anatomical lesion had not at least reached its maximum, namely, the stage of fatty metamorphosis. M. Ch. Robin, it is true, declares that he has never met with this change, but his views are opposed by men whose opinions in microscopical matters is law in science. I need only mention Professors Virchow (of Berlin), Friedreich (of Heidelberg), and Lebert (of Breslau), who believe, as do also Professor Cruveilhier and M. Duchenne (de Boulogne), that progressive muscular atrophy is anatomically characterised by the diminution in size of the muscular bundles (the transverse and longitudinal striæ of which are seen to disappear at a more advanced stage), and, lastly, by the production of granulations, which ultimately become fatty. I declare myself incompetent to decide the question. But whether these granulations be fatty or not, the point, however interesting it may be in pathological histology, is of little importance in a clinical point of view. It would be of greater use to us if we could know whether the muscular lesion is primary, or whether it is dependent on a lesion of the nervous system, either of the cerebro-spinal centres, or, as it has been stated, of the anterior roots of the spinal cord. You are aware, gentlemen, that in his memoir on *Progressive Atrophic Muscular Paralysis*, read at the Academy of Medicine in March 1858, and published in the "Archives of Medicine" in May of the same year, Professor Cruveilhier adopted the conclusion that this motor paralysis, which was sometimes local and sometimes general, and coincided with the complete retention of sensation and intelligence, was due to atrophy of the anterior roots of the spinal nerves. My honoured colleague quoted in support of his view the case of a man named Lecomte, which had been already given in detail in a memoir by Dr. Aran,[1] and

[1] Archives de Médecine, Sept. 1850.

has been since reproduced in the work of Dr. Duchenne (de Boulogne).[1] There was, in that case, marked atrophy of the anterior roots of the spinal cord, in the cervical region chiefly; and in the case which suggested the present lecture, there was found atrophy of the same parts. From other cases, however, in which these roots are said to have been in a perfectly normal condition, one may be led to believe that this peculiar lesion was by no means the primary cause of the progressive muscular atrophy.

As to a lesion of the nervous centres, the integrity of the intellectual functions, the absence of all symptoms during life of paralysis proper, and after death the absence of anatomical changes in the spinal cord and the brain, prove conclusively that the great centres of innervation are not in the least involved in this complaint. And yet I have told you that M. Sappey thought he had found in my patient a diminution in size of the anterior columns of the cord.

Even though progressive muscular atrophy belong to the class of neuroses, as I believe and admit, the morbid process which characterizes it still begins primarily in those muscles themselves which are involved, and in their intimate elements. However interesting it may be for a physician to acquire precise notions as to the proximate cause of a disease, what he requires above all, and what is to him of immediate use, and he cannot dispense with, is a full knowledge of the symptoms and a correct appreciation of the characters by means of which alone he will be enabled to diagnose the disease. Let us, therefore, enquire what are the *symptoms of progressive muscular atrophy*. They have such a peculiar physiognomy that, when they have been once observed with attention, they can be very rarely mistaken. The first sign of disease of which the patient complains is a diminution of muscular strength, at first confined to one limb only, and increased by cold and by exercise—the difficulty which he has in executing a movement sometimes increasing to such a degree that he is perfectly incapable of performing it. This weakness, which is local at first, confined to a single limb, and even to a portion of one limb, and affecting certain movements only, is generally attended with cramps (*subsultus tendinum*), whilst the diseased muscles are often the seat of fibrillary contractions. I say *often* only, because one must not think that these fibrillary contractions are a sympton *sine quâ non* of progressive muscular atrophy, and constitute one of its fundamental characters: for, on the one hand, it is not rare to find that they are completely absent during the whole course of the complaint, and, on the other hand, they are observed in other muscular affections which are very different from the one under consideration. They are analogous to the contractions which are noticed in diseases of

[1] "De l'Électrisation localisée." Paris, 2e édit., 1861, p. 437.

the spinal cord, and, like them, they are independent of the will, occur spontaneously, but are never so violent and so repeated as when the muscles in which they show themselves are excited, either by being percussed, compressed, pinched, or maintained in a state of forcible contraction, or when they are galvanized. They last a very short time, and sometimes follow one another with such rapidity, and such frequency, that the affected muscles seem to be in a state of constant action: at other times, on the contrary, they are so very rare that one must watch the moment when they occur, and even irritate the muscles in order to produce them. When they do occur, they give rise to an appearance of very fine whipcords, which are alternately tightened and relaxed under the skin, with extreme rapidity: at other times the movements are slight and of a vermicular character. As they are unattended with pain, and are involuntary, they are often unperceived by the patient, although they are sometimes spoken of by some as a very slight quivering. When these fibrillary contractions affect a muscular bundle of pretty large size, they give rise to very perceptible convulsive movements, chiefly in the limbs; and patients state that their fingers are alternately flexed and extended suddenly, independently of their will.[1]

This sensation of quivering is not the only one, according to Dr. Duchenne (de Boulogne) which is mentioned by individuals suffering from progressive muscular atrophy. In a pretty advanced stage of the disease, they complain of a sense of cold in the atrophied limb, consequent on a real lowering of its

[1] [That these fibrillary contractions are not a constant symptom is sufficiently shown by the fact of their having never been noticed by competent observers. Thus, Dr. Meryon states that he has never seen them, and adds: "On the contrary, the singular absence of every indication of nervous disturbance has obtruded itself on my mind." ("On Paralysis," p. 207). Dr. Duchenne (de Boulogne), on the other hand, declares that they were absent in a full fifth of his cases. In fifteen cases which have come under my own notice, the absence of all quivering and of all fibrillary contraction was ascertained in two instances, both during the time the patient was under observation as well as anteriorly also, at least as far as could be made out by close questioning. In all the rest this phenomenon was well marked. The conjecture now suggests itself, whether these involuntary contractions of individual muscular fibres may not manifest themselves only when the anterior roots of nerves have become affected, or the anterior columns of the spinal cord have been implicated in the disease? Their absence would be thus explained in Dr. Meryon's cases, in some of which a post-mortem examination revealed no nervous change; whilst in the case of the showman Lecomte (Professor Cruveilhier's celebrated case), in whom these fibrillary contractions were so very marked, the anterior roots of the nerves which supplied the atrophied muscles were found reduced in bulk. In a patient under the care of Prof. Trousseau, who died at No. 23 in St. Agnes Ward, and whose case is related a few pages farther on, the sensation of quivering was highly marked during life, and after death the anterior roots of the cervical and dorsal nerves were found in an atrophied condition.—Ed.]

temperature which can be appreciated by the physician. The capillary circulation has by this time become less active, whilst the cutaneous veins enlarge, and the skin gets livid under the influence of cold. The muscular weakness, which is the first striking symptom of progressive muscular atrophy, very rarely sets in suddenly; and even when this seems to have been the case, the accuracy of the patient's statement might very well be questioned. Nearly always, not to say always, the disorders of locomotion manifest themselves slowly and gradually. At the outset, as I told you a moment ago, this weakness is localized in and restricted to a limb, or even a part of a limb, interfering with some movements more than with others, increasing under the influence of cold or of fatigue to such a degree as to take away all motor power. By degrees the affection becomes general, and involves the whole of the limb—not all its muscles in an equal degree, however, and even sparing some of them entirely. The opposite limb is in its turn attacked, and, at last, the whole muscular system of the life of relation is more or less generally implicated.

According to Dr. Duchenne, this weakness is not due to deficient nervous excitability, but is the result of changes in those muscles, the fibres of which are more or less destroyed, and which consequently lose the power of performing movements. Voluntary muscular contractility remains normal unto the end, even in the most advanced stage of the disease, in those fasciculi which have not undergone a morbid change. This is an all-important fact, which has been and could only be brought to light by means of localized faradization. I hasten to add that the discovery of this fact is solely due to Dr. Duchenne (de Boulogne).

No one is more disposed than I am, gentlemen, to give Dr. Duchenne his due, and to declare that to no physician is the study of diseases of the nervous system more indebted than to him for real progress, but I cannot completely endorse his opinion touching progressive muscular atrophy. I read to you, in the beginning of this lecture, the details of the microscopical examination made by Dr. C. Robin, and you may remember that there still remained in that case a good many muscular fibres which were apparently sound, whilst others were already altered, and others again were so very discoloured, and so deeply modified, that one could conceive that they had lost all contractile property. You may remember also that Dr. Duchenne himself, who honoured us with his presence during our round, had shown that most of the muscles of the arm and forearm still contracted under the influence of electricity, whilst the patient could not voluntarily move his hands or his forearms. One could but suppose, therefore, that previous to any anatomical change

which seemed not to exist then, the peripheral extremities of the nerves had undergone a modification, in consequence of which they had lost the power of rousing muscular contraction. A loss of excitability of the peripheral extremities of nerves would therefore precede the degeneration of the muscular fibres, a fact perfectly in accordance with pathological physiology.

The difficulty felt in the performance of movements generally coincides with very marked thinning of the diseased parts. I say generally, because cases have occurred (and Dr. Duchenne has at least recorded a remarkable instance of the kind) in which muscular atrophy coexisted with a considerable degree of plumpness, and was only characterized by a diminution of motor power. The emaciation presents, besides, a very characteristic feature. It is only seen in those parts which correspond to the diseased muscles, while the rest retains its normal size; and it differs, therefore, from the general emaciation which obtains in individuals exhausted by prolonged suffering, or which is sequential to paralysis, even when localized, as in the case of lead-palsy. The destruction of the muscular masses, which is the cause of these alterations of shape, also produces changes in the attitude of the limbs and trunk during muscular rest, in consequence of the loss of equilibrium between antagonistic muscles. Lastly, apart from motor weakness, there is also inability to coordinate voluntary movements, as the diseased muscles can no longer act in concert with the rest.

I shall presently revert to these peculiarities; but a most curious one, to which I am desirous of first drawing your attention, is the favourite seat of progressive muscular atrophy, at its onset, in the upper extremities.

Usually—at least 9 times out of 11, according to an analysis of cases made by Aran—the disease primarily attacks the upper limbs, and more especially the right limb, 7 times out of 11. Its seat is still more localized, for the muscles of the hand are the first to be involved—those of the thenar eminence in the very beginning, those of the hypothenar next, and the interossei afterwards. You must not think, however, that all the muscles of the diseased region, or even all the fasciculi of a single muscle, are simultaneously affected. This is far from being always the case, for by the side of atrophied muscles, there may be others in the same region, their congeners even, which are untouched and act in the place of the former, as these are necessarily unable to discharge their functions. Again, there may be in the same muscle, by the side of fasciculi which have undergone a morbid change of structure, other fasciculi which may be called into contraction by means of faradization, and which must therefore be made up of healthy fibres. Thus,

in the case of a patient lying in bed 23, St. Agnes Ward, which I shall presently relate to you, the atrophy commenced in the left deltoid muscle, and yet, at the end of three years and a half, the posterior fasciculi of that muscle were still in a nearly normal condition. They contracted in obedience to the will and under the influence of electricity, whilst after the patient's death, they were found to be of normal colour and size, and under the microscope they exhibited very regular transverse striæ. The fibres of the anterior and middle fasciculi, on the contrary, either showed no transverse striæ at all, or only in spots here and there; or even, as in the ultimate stage of the degeneration, the sarcolemma only contained extremely fine granulations, mixed up with fat-cells in greater or less numbers.

This is the rule, but there are exceptions to it, for Dr. Duchenne (de Boulogne) has pretty frequently seen the disease commence in the muscles of the trunk. Thus, he saw it once begin in the sacro-lumbales muscles; in another case, the pectorales, trapezii, and latissimi dorsi were destroyed before the upper limbs became involved; and then a large portion of the biceps in the arm, and the supinator longus in the forearm, were affected, whilst the motor muscles of the hand escaped entirely. In another case, again, the muscles of the trunk, the pectorales, trapezii, rhomboidei, latissimi dorsi, serrati magni, some muscles of the lower limbs, among others the flexors of the leg, were atrophied, whilst in the upper limbs the supinatores longi were alone destroyed; in two cases the lower limbs were the first involved. Lastly, in a case which I had the opportunity of seeing with Dr. Duchenne (de Boulogne), and which he has published in his treatise on *Localized Electrisation*, the disease became general in less than two years, and ran a most irregular course.

The patient was a Spaniard, aged 32, who had come from Barcelona to Paris. The motor muscles of the right hand were the first to waste away, and after them the flexors of the left foot. The left hand atrophied next, and after it the flexors of the right foot and those of both thighs. Lastly, the atrophy extended in a variable degree and in the following order—to the biceps, the deltoidei, the muscles of the trunk, those of the neck, and those of the face. At the time when these notes were taken, the diaphragm and the muscles of deglutition were so seriously involved, as to threaten the patient with the risk of dying of starvation or of asphyxia.

At the close of the year 1860, a gentleman, practising in the South of France, sent me a lady, aged 30, who had been paralysed for several years past. She was remarkably plump and fresh-looking, and I only saw that she had lost all muscular

power, but could not find out the cause of it. Having asked Dr. Duchenne to join me in consultation, I must declare that before he had asked the lady four questions, he had recognised, and made me recognise, with the greatest facility, progressive muscular atrophy, hidden under heaps of fat. What had contributed to deceive me, was the fact that the lady, who was an excellent musician, tried to console herself of her sad infirmity by playing the piano, so that she had lost none of the strength and precision of the movements of her hand and forearm. In her case the deltoidei, rhomboidei, serrati magni, sacro-lumbales, psoæ and iliaci muscles, were more particularly diseased.

I repeat, gentlemen, these are exceptions, which do not in the least invalidate the general law, that muscular atrophy first shows itself in the upper extremities. The course of the disease has been carefully studied, and it has been ascertained that after the muscles of the hand, especially those of the thenar and hypothenar eminences, have been involved, the interossei, and the flexors and extensors of the fingers, and in some cases the muscular masses of the posterior region of the forearm, atrophy in their turn.

The disease, being thus restricted to these localities, may remain stationary and not spread beyond these limits for several years; but when it passes them, it affects almost simultaneously the muscles of the arm and of the trunk, although in every case only partially and very irregularly. In the arms, the biceps wastes and then the deltoid—in some cases the former before the latter; in others, on the contrary, the reverse obtains. The triceps is the last to undergo change. According to Dr. Duchenne, the course of the disease is generally the following: the trapezius disappears first, but it is a remarkable fact that its lower portion alone does so; whilst its clavicular portion is, on the contrary, the last involved of all the muscles of the trunk and neck. Next in succession come the pectorales, the latissimi dorsi, rhomboidei, levatores anguli scapulæ, the extensors and flexors of the head, the sacro-lumbales, and the abdominal muscles. In nearly every case Dr. Duchenne has found that, at this period of the disease, the muscles of respiration, of deglutition, and of the face become involved.

If, in very rare cases, muscular atrophy shows itself first in the lower limbs, in general the muscles of those regions undergo transformation only after those of the upper limbs and trunk have been to a great extent destroyed. The disease seems to concentrate itself in the flexors of the foot on the leg, and those of the thigh on the pelvis, the remaining muscles undergoing change in the long run only.

The disease never shows itself at once in the two sides of the body, but when it has attacked a certain group of muscles, their

homologues are not long before they are similarly affected, previous to the extension of the disease to other regions.

The modifications in the shape of parts which have lost their muscles, the changes in the attitude of a limb and the trunk, consequent on the destruction of the muscles, are characteristic of and special to progressive muscular atrophy. I mentioned to you just now the emaciation which accompanies the weakness of the patient's movements, and the loss of freedom in their performance, of which he complains; and I stated that the emaciation was by no means like the one which follows long-continued and exhausting diseases, or which obtains in paralysis, when the parts which have lost the power of moving diminish in size. In such cases, even when the paralysis is confined to a certain group of muscles, as in lead-palsy, the emaciation is uniform, while it is merely partial in muscular atrophy—so much so, indeed, that by the side of parts almost deprived of all muscular substance, others may be seen which have preserved their regular shape, and the prominence of which contrasts with the depression of the former.

This characteristic alteration of shape and these contrasts may affect all the various regions of the body, or they may be confined to more or less limited areas, and present an infinite variety of aspect and seat. Thus, an individual whose chest-walls are perfectly bony from the atrophy of his pectorales muscles, and whose scapula and its bony eminences stand out in relief through the disappearance of the fleshy masses of his back and of his trapezius and rhomboidei, may have arms which are still strong and with well-developed muscles, a face with perfectly regular features and of its usual plumpness. In another case, in which the disease has not spread beyond the upper extremities, you will be surprised to find that the hand and forearm have wasted considerably, whilst the muscles of the arm have undergone no change. In another case, again, the atrophy being incipient only, and having attacked the hand alone, the thenar eminence has disappeared, and in its place a hollow is seen, caused by the disappearance of the superficial and deep layers of muscles; and later, deepening of the interosseous spaces is noticed, consequent on the degeneration of the muscles which filled them. I shall not pursue this analysis further, for the best descriptions cannot give an accurate idea of these alterations of shape which are special to atrophy, and which need be seen but once in order to enable one to recognise the disease at first sight.[1]

[1] [Atrophy of the interossei muscles of the hand produces that peculiar deformity which has been termed by Dr. Duchenne (de Boulogne) the "*main en griffe*," the bird's-claw hand, and which results from the first phalanges being extended while the two last are flexed. The deformity is considerably

There is one point, however, to which Dr. Duchenne (de Boulogne) has called attention, and which I must not omit—namely, that this alteration of shape, which is a pathognomonic feature of atrophy, may be absent in some cases, even when a great many muscles are entirely atrophied, from its being masked by a considerable degree of stoutness. I mentioned to you just now a remarkable instance of the kind, and another is recorded by Dr. Duchenne—namely, that of a man afflicted with progressive muscular atrophy, localized on each side in the trapezii, rhomboidei, latissimi dorsi, and serrati magni, coinciding with extreme obesity. The case is so interesting on many counts, that I must request permission to quote it *in extenso*: "M. R——, of Aix, in Provence, aged 22, tall, extremely stout, and of a robust constitution and sanguine temperament, has had no other ailment but the one for which he is now applying for advice. His great-grandfather, his grandfather, and his father, all of them *eldest sons*, were attacked with the same complaint—the first between 20 and 22 years of age, the second when 24 years old, and the third at the age of 17. In all of them the disease showed itself successively in the muscles which move the shoulder, next in those of the arm, and lastly in the flexors of the thigh on the pelvis, and those of the foot on the leg. M. R—— became affected at the age of 17. Until then he had been rather thin; but from the age of 18, he grew gradually stouter, so much so as to be exempted from the conscription on account of obesity. From the time that this obesity set in (which likewise showed itself in the case of the members of his family who laboured under this muscular affection), there came on weakness of some movements. First, the act of raising the arm, or of carrying his hand to his head, became more and more painful. During this

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exaggerated when the patient attempts to open his hand, for the extensor digitorum (which extends the first phalanx only) is no longer counterbalanced and regulated by the interossei, which, according to Duchenne, besides adducting or abducting the fingers, subserve the important purpose of flexing the first phalanges and extending the two last. At a more advanced stage of the disease, when the flexor and extensor muscles of the hand have wasted away, with the other muscles of the forearm, the deformity of the hand disappears, and the limb assumes a skeleton-like aspect.

It is important to ascertain whether the intercostal muscles are atrophied or not; for, in the former case, the patient runs the risk of dying of asphyxia on the supervention of the least bronchitic attack, so that it becomes imperative to guard him against any such contingency. When these muscles are atrophied, the patient, according to Duchenne, can neither cry nor sing, although there is no aphonia, so long as the diaphragm is not implicated. The voice is weak, however, and the patient speaks in broken sentences only, stopping between his words to draw breath. When he inspires, the lower part of his chest moves alone, while the two upper thirds remain immovable; and from his lungs taking in very little air, he cannot expel a sufficiently large column of air to blow a candle out.]—Ed.

movement the scapulæ were extremely prominent, and their inferior angle, instead of moving outwards and forwards, moved backwards. These phenomena became gradually more and more marked, until the present date. M. R——, for the last twelvemonth, has complained of some fatigue in walking, and especially in going up a staircase. He has never felt any pain, and has not had articular or muscular rheumatism, or any syphilitic affection.

The disease, which has for the last three generations attacked the eldest sons in his family, has until now remained a mystery. Although he has already lost to a great degree the power of raising his arms, he still hopes that he may escape the fate of his predecessors, because he believes that the disease is, in his case, limited to the muscles which serve to raise the arm, and grounds his belief on the development of his limbs and the soft parts of his trunk.

On testing the condition of his muscles, however, by electricity, a large portion of the pectorales were found to have disappeared, and no traces could be detected of the trapezii, rhomboidei, latissimi dorsi, and supinatores longi. The other muscles were well developed, and contracted very powerfully when galvanized. M. R—— could not raise his arms above the horizontal line, and even then he had to make very great efforts. During that movement the spinal border of the scapula moved away from the thorax, whilst its inferior angle got nearer the median line. In that position the scapula formed a triangle, the apex of which was at the inner angle of the bone, and the base was formed by its axillary border. The atrophy of the other muscles did not seem to interfere much with his movements. Lastly, fibrillary contraction was not perceptible anywhere, and the patient declared that he never felt any quivering of his muscles. His general health was excellent. On merely looking at M. R——, one would not indeed suspect that atrophy had already destroyed many of his muscles. His chest is plump and well developed; the posterior aspect of his trunk looks normal and well nourished, when his arms hang alongside of it; and yet, on testing his muscles by galvanism, the trapezii, rhomboidei, and latissimi dorsi are found to be missing. As these muscles are only of secondary importance, their atrophy would not be suspected if the serrati magni were not involved. M. R—— was surprised to hear, therefore, that all these muscles had degenerated, and especially that the supinatores longi were destroyed, while his arms possessed vigorous muscles. He knew that he was seized with the complaint which was hereditary in his family, from the difficulty which he had from the beginning in raising his arms, owing to the absence of the serrati magni. The special deformity, which

is observed when the arm is raised, from the want of co-operation of the serratus magnus, and which is pathognomonic of paralysis of that muscle, is the only sign which at first sight points to the existence of muscular degeneration.

Cases of this kind are too exceptional to detract from the value of alteration of shape as a pathognomonic sign of progressive muscular atrophy. In proportion as the disease makes progress, this character becomes more and more marked, and there comes a time when the skeleton-like aspect of the greater portion of the body contrasts with the plumpness of the face.

The disease derives a still more special physiognomy from the changes which occur in the attitude of the limbs and the trunk in the state of rest, and from the disorders of locomotion during the performance of voluntary movements. These phenomena have been accurately studied and analysed by the author of the treatise on *Localized Electrisation*:—

"The attitude of the limbs during muscular rest depends on the tonicity of the muscles which move them. But there is no muscle which is not antagonized by another. If, therefore, one of the antagonistic muscles happens to be weakened or destroyed by atrophy, the equilibrium of the tonic forces, on which depends the normal attitude of the limbs, is disturbed, and the limbs are necessarily drawn in the line of the predominating tonic force, namely, of the predominating muscle or muscular bundle. If the mechanism of these faulty attitudes be well understood (and this may be done by studying the separate action of individual muscles and even of muscular bundles), it will be easy to deduce from it the kind of signs which constitute the principal characters of partial atrophies."[1]

The functional disorders, which occur during the performance of voluntary movements, affect, some of them, the movement special to a muscle or a portion of a muscle, and others the co-ordination of the diseased muscles; for every movement, in order to be regular, not only requires that one or several muscles should contract, but also that other muscles, which do not directly concur in the production of the principal movement, should come into play, in order to steady, regulate, and moderate that movement. The patients can supply the place of some of the muscles which they have lost, by instinctively contracting their congeners: as when, for instance, in the absence of the biceps, the forearm is flexed by means of the muscles which are inserted into the epitrochlea, and especially by means of the pronator radii teres. These supplementary movements are very irregular, it is true; but there are muscles which have no

[1] "De l'Électrisation localisée, et de son Application à la Pathologie et à la Thérapeutique." 2e édition, Paris, 1861, p. 468.

homologues, and then the movements which they used to perform become impossible; and if the patient attempts to execute them perfectly, contrary movements will result, through the antagonistic muscles contracting by themselves. I cannot too strongly recommend to you, gentlemen, to read in Dr. Duchenne's treatise the interesting details into which he has entered on this point, and which it would take me too much time to go into here.

However advanced the destruction of the atrophied muscles, and however general the disease may be, phenomena indicating a general disturbance of the system are absolutely wanting. The appetite remains good, and digestion is perfectly regular. Yet, when the muscles of mastication and deglutition become in their turn involved, these acts are more or less impeded in consequence. The lower jaw is depressed with an effort only (for it is the depressors of the jaw which are in general affected), and there may even come a time when, owing to the complete destruction of these muscles, the mouth cannot be opened, and the patient has extreme difficulty in feeding himself. Deglutition is generally then accomplished with difficulty, and attended with a copious flow of saliva. I need not tell you what a grave complication this will be in the patient's condition, in proportion to the degree of interference with the mechanism of deglutition. The amount of food taken being insufficient, the patient may die from gradual starvation.[1]

[1] [It rarely happens that the tongue is affected in progressive muscular atrophy, but, although rare, this complication is met with in some cases. Of three instances of this disease related by Romberg (*loc. cit.* vol. ii. p. 373), the tongue was involved in one, "vibrations manifesting themselves in its muscular fasciculi, by which articulation and deglutition were much impaired, although the tongue continued freely moveable in all directions." I have myself seen wasting of the tongue in 2 out of 15 cases of well-characterized progressive muscular atrophy which have come under my observation. The first of these has already been published (at p. 140 of Part I. of this work); and the subject of the other, a woman at present under my care at the Hospital for the Paralysed and Epileptic, is afflicted with general muscular atrophy involving most of the muscles of the four extremities, some muscles of the trunk, as well as those of the lips, tongue, and soft palate. The wasting of the muscles of the lower limbs is such as to prevent her from getting about, except with very considerable difficulty, even when supported under the arms; while, in consequence of the atrophy of the tongue, she labours under such difficulty of deglutition, that she is only kept alive by being fed with the greatest care. This is the only instance which I have yet seen, I may add in passing, of progressive muscular atrophy becoming general in a female, individuals of that sex suffering, as a rule, from localized forms of the complaint.

Dr. Duchenne (de Boulogne), an excellent authority on this point, states that he has met with wasting of the tongue in progressive muscular atrophy only 15 times out of 159 instances of the disease which he had seen up to the time of his making that statement ("Traité de l'Élect." 2e éd. p. 644); and he insists on the fact that this complication supervenes late in the course of the disease, and is unattended with paralysis until the tongue is considerably wasted.

As the rectum and bladder are never affected, the fæces and urine are normally expelled, at will and with perfect freedom,

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Cases in which there is paralysis of the tongue, unaccompanied by wasting, together with palsy of the lips, tongue, and soft palate, Dr. Duchenne groups together as instances of a distinct affection, for which he has suggested the name of *glosso-laryngeal paralysis*. Since the publication of the first part of this work, in which this subject is so ably treated by Prof. Trousseau, I have had an opportunity of seeing a case of this singular affection in a private patient of my friend and colleague Dr. Ramskill, who was kind enough to ask me to see her in consultation with him. Such cases are so very uncommon that I am tempted to insert this one here:—

Epileptiform Convulsions.—Persistent Weakness and Anæsthesia of the Left Arm.—Progressive Paralysis of the Lips, Tongue, and Soft Palate.

Mrs. B——, aged 38; tall, thin, the mother of one child; no arcus senilis; hair slightly grey; previous health very good; no rheumatic fever.

Present complaint dates from about the middle of 1863. For three or four weeks previously she had complained of numbness in the forefinger of the left hand. One evening, at a party which she was giving, she suddenly felt a strange sensation in her left hand, which rapidly spread to the left cheek, and was accompanied by complete anæsthesia of the left arm, so complete that she did not know that she had an arm without looking at it. She left the drawing-room, but on reaching an outer apartment, she dropped down senseless, and was seized with violent epileptiform convulsions, affecting both sides of the body equally, the face and limbs, and attended with foaming at the mouth, blueness of the lips, &c. For two or three hours after this seizure, she remained in a comatose condition. Two days subsequently she had a second attack of a similar nature. For fully three months afterwards she suffered from intense pain in the head, constantly present, felt chiefly in the occipital region, and from there extending to the vertex. She always kept her hand pressed to the back of her head. She complained also of a sensation of blood rushing to her head, of singing in her ears, and is half inclined to state positively that the singing noise was heard in the left ear alone. During that period the anæsthesia of her left arm diminished, but did not entirely disappear. Her hand was half-closed, and although the motility of the arm itself was not affected, she could not use her hand on account of the attitude of the fingers and the numbness of the hand. For the first year she was subject to convulsive seizures, recurring at variable intervals of from two or three days to a fortnight, never exceeding three weeks. She did not lose consciousness at every seizure, which always began with stiffness and rigidity of the left hand.

During the second year the fits were as frequent as in the first, but were perhaps less severe. On one occasion she had as many as 200 fits in the course of two or three days, and then none for a month.

In February 1865 she had a fit, followed for four days afterwards by complete loss of power in the *right arm*; the sensibility of the limb was merely diminished, not abolished. During the first six months of her complaint, she had been troubled with violent sickness, even when taking no food, and on this occasion this troublesome symptom recurred again. For four or five months after this attack, she became subject to twitchings of the muscles of the right cheek, and oscillations of the right eye, coming on in paroxysms every three or four minutes. It was not until the following August that she had an epileptiform seizure, preceded by pain and stiffness in the left arm, and jerking of the limb. Since that time she has complained of peculiar spasms of the thumb and index-finger of the left hand. From August to October 1865, she was free from fits; but in the latter month these returned again, beginning always with involuntary closure of the right hand. Since October the fits have recurred, at very variable intervals, every day for two or three days—then once a week, or once a fortnight. Each fit is ushered in by a convulsive twitching of the

unless the abdominal muscles should be atrophied, in which case their expulsion is attended with difficulty.

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muscles of the lips. Sometimes, however, she has the twitching but no fit, although she declares that she can tell the difference between the two sensations. The fit presents the well-known characters of epileptic seizures; the patient falls down, foams at the mouth, is convulsed generally, &c.

In addition to the above seizures, Mrs. B—— labours under another affection —namely, progressive paralysis of the lips, tongue, and soft palate. She cannot positively state when the first symptoms of this peculiar disease manifested themselves, and in what order they appeared. She declares, however, that the first symptom which attracted her attention was a dribbling of saliva, which was especially troublesome whenever she talked. This set in, she thinks, about the same time nearly as her epileptiform seizures, or, at the least, very shortly afterwards. She had not at first any difficulty in swallowing solids or liquids, or in clearly enunciating her words. These latter symptoms did not show themselves till about a year after the dribbling had commenced, that is, about twelve or eighteen months ago. They came on very gradually, and she clipped her words for three or four weeks before her difficulty of articulation was noticed by strangers with whom she conversed. In February 1865 (after the fit which was followed by motor paralysis of the right arm) she completely lost all power of articulating, and this persisted for three months. She remembered words and the names of things well, knew perfectly what she wanted to say, but was incapable of articulating. On no other occasion has she again completely lost the power of articulation, but her speech, after she recovered it, became gradually more and more thick and embarrassed; while the difficulty in swallowing has, by degrees, increased to such a point that she has for some months past been obliged to use her fingers to push the bolus of food back from the tongue into the pharynx, and to get the food out from between the cheeks and teeth.

I saw Mrs. B—— for the first time in January 1866, and noted down the following facts. Her intellect is unaffected, her memory excellent, her hearing and sight good; there is no ptosis, no strabismus, no headache. Her eyes look intelligent, but the lower part of her face is heavy and expressionless. The lower jaw hangs down a little, and no lateral, grinding movements are possible. The elevation of the lower jaw is so deficient in force, and its muscles so weak, that the patient cannot tear or bite with her incisors. The lips are always partially open, and through them the tongue can be seen lying on the floor of the mouth, looking large and, as it were, swollen. In consequence of the constant dribbling of saliva through the half-opened lips, especially when she talks or inclines her head slightly forwards, the patient is obliged to keep a handkerchief to her mouth.

The sensibility of the cheeks, lips, tongue, and mucous lining of the mouth is normal. Taste is unaffected. The orbicularis oris is imperfectly paralysed of movement, for the lips cannot be closely approximated together and closed, so as to prevent the escape of air from the mouth when the patient is asked to inflate her cheeks. She cannot purse up her mouth or whistle, and when she drinks, liquids have a tendency to run out of the corners of the mouth.

The motility of the *tongue* is considerably impaired; in size the organ looks larger than natural, and therefore presents no appearance of wasting. It cannot, however, be protruded beyond the edges of the lower teeth, and even then with extreme slowness and manifest exertion. It cannot be moved from side to side, or its tip raised upwards, or its sides curved inwards so as to make the centre of the organ hollow. There is considerable difficulty of swallowing; liquids are taken in very small mouthfuls at a time, and swallowed slowly and carefully; they often get into the larynx, causing fits of coughing, and sometimes come back through the nose. Solids have to be pushed back into the pharynx with the fingers in the first stage of deglutition. On looking down into the mouth, the soft palate is seen to hang in a semi-paralysed condition;

The breathing is regular, until the time when the respiratory muscles are involved, but this generally occurs in a pretty advanced stage of the disease. When the diaphragm is affected, phonation is impaired, and becomes considerably more so when the expiratory muscles atrophy. Were these to disappear completely, simultaneously with the diaphragm, asphyxia would result from the complete inability to breathe; but before the lesions reach that degree, they can cause the most serious complications, in the shape of intercurrent diseases of the respiratory apparatus, and thus prove an indirect cause of death. If the patient be attacked with bronchitis, for example, the mucus secreted in the bronchial ramifications can no longer be expelled, and their accumulation in those tubes will bring on asphyxia more or less rapidly.

A patient, whom you may have seen at No. 23 in St. Agnes Ward, died in this way. He was a cabinet-designer, and 46 years old. Three years and a half ago he first noticed that the strength of his left shoulder failed him, and that it was less easily moved. He felt, at the same time, in his deltoid, creeping and quivering sensations, which were the fibrillary movements characteristic of incipient muscular atrophy. The shoulder shortly became thinner, and then perfectly identical symptoms showed themselves in succession in the biceps, in most

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during deep inspiration it rises imperfectly only; when tickled, it rises better through a reflex action. The voice is feeble at times, and very indistinct; certain letters are pronounced very well, namely the vowels *a* and *i*; others, such as the labials *b*, *p*, *t*, *d*, are enunciated indifferently well; while the linguals, *l*, *g*, and *z*, are very badly uttered, and *s* has the sound of *th*.

Respiration is regular, but there is imperfect lateral dilatation of the chest during deep breathing, the upward movement being performed better. The deficiency of the respiratory capacity is well exemplified by the difficulty which the patient has in blowing out a lighted candle held about a foot from her. Her appetite is capricious, but digestion is easy. Bowels regular. Bladder rather irritable or weak; has frequent desire to pass water; there has never been incontinence of urine. Menstruation regular, but insufficient. Heart-sounds normal.

With regard to the limbs, the lower ones have never been affected in any way. The right arm is also well in every respect. The left is, however, weak, and the patient cannot squeeze with any degree of force. There is considerable anæsthesia of the hand; the patient cannot pick up a pin unless she looks at it, and never trusts herself to carry a glass to her mouth with that hand alone. There is not the slightest trace of wasting in the muscles of the hand or arm.

I have lately seen Mrs. B—— again (October 1866), and ascertained that there is no wasting whatever of the tongue, or any muscle of the hands, thus showing very clearly that the case is not one of Cruveilhier's disease commencing in the tongue. The organ contracts very well under the influence of faradization directly applied to its muscles. The paralysis is not of greater degree than before, and there have been no epileptic seizures lately—merely spasmodic twitchings of the lips, which the patient watches herself in a glass. The left hand feels weak still, and there is the same inability as before to use that hand, particularly the index-finger.]—Ed.

of the muscles of the forearm, and in those of the thenar and hypothenar eminences. Soon afterwards the right shoulder, arm, forearm, and hand were similarly affected. Note that in this case the disease spread from above downwards, from the shoulder to the hand, whilst the reverse usually obtains.

At the end of eighteen months the patient had to give up his occupation, and for the last two years he had only been engaged as a messenger, when he decided on being admitted into one of my wards, on October 2, 1863. You could then see that both his shoulders were atrophied, and that his humeri, being no longer supported by the deltoid muscles, dropped from the glenoid cavity of the scapula. His upper limbs hung alongside of his trunk, and when he wanted to feed himself, he knelt by a small table, rested on it his forearms (which were by that means flexed, in the absence of his wasted biceps), and took hold, with both his hands, of the pieces which he wanted to carry to his mouth, making the best use he could of the muscles or of the remnants of muscles which he still possessed. His chest-walls were fleshless, and it was evident that his pectoral and intercostal muscles were wasting away. The muscles of his abdomen and lower limbs were intact.

All the functions of vegetative life were perfectly performed; yet I told you beforehand, that the imminent danger which this patient ran was from the possibility of his having a chest-affection, for he had an habitual small dry cough. On January 16, he became suddenly feverish, had difficulty of breathing, and died two days afterwards from this dyspnœa, which I vainly tried to combat. I subsequently found general congestion of, and miliary granulations in, both his lungs. You saw the pathological specimens which my clinical assistant, Dr. Peter, had prepared. The deltoid muscles, the biceps, coraco-brachiales, the superficial and deep flexors of the fingers, &c., the muscles of the thenar and hypothenar eminences, the interossei and lumbricales, were more or less considerably atrophied; whilst the triceps brachialis, the palmaris, and anconeus were untouched by disease, and their normal volume and their red colour contrasted with the slenderness and the yellowish discoloration of the wasted muscles. The pectorales minores and majores were partially atrophied, and the intercostales very considerably so.

The degree of atrophy did not merely vary in the limbs, but also in various portions of the same muscle. Thus the muscles of the left arm were markedly more atrophied than their homologues on the opposite side, and the posterior bundles of the deltoids were normal, whilst their anterior and middle bundles were deeply altered.

You could ascertain, by comparing them with those of a

healthy cord, how far the anterior roots of the cervical and dorsal nerves had atrophied, whilst the lumbar nerves and those of the cauda equina were of normal size. The roots of the cervical nerves were those especially affected, and more markedly so on the left side. Some of them, particularly those which go to form the brachial plexus, were reduced to one or two extremely fine threads. The circumflex, median, ulnar, and radial nerves were of somewhat diminutive size.

I was anxious that not only the structure of the anterior roots and of the nerves should be carefully examined, but also that the condition of the capillary bloodvessels distributed to the atrophied muscles should be ascertained. Now, Dr. Peter found that the nerve-tubes of the most diseased roots and nerves had diminished in number and size, and that the diminution in size was due to a considerable reduction in the amount of nerve-substance. The nerve-substance of some tubes had become finely granular, while it had completely disappeared from others, as well as the cylinder-axis; and there remaining nothing but the sheath, the tube had a remarkably constricted appearance.

(In the examination, a comparison was instituted between the above tubes and those of healthy roots and nerves.)

The changes of the nerve-elements consisted, therefore, in a diminution, a granulation, or a complete disappearance of the nerve-substance, with retention of the neurilemma. We shall presently see that these changes are perfectly analogous to those of the muscles. No appreciable lesion of the capillary vessels could be discovered.

As to the muscular fibres, they presented various degrees of alteration. They had diminished in size, and while, in some fibres, the transverse striæ had merely become more rare, and fat-globules were seen in pretty large numbers, in other fibres the striæ had completely disappeared, and in their stead were only seen very fine granulations. Hence, then, a microscopical examination revealed that the sarcolemma of the muscular fibres was preserved as the neurilemma of the nerves was, and that there was a diminution or a granular degeneration of the proper substance of both tissues, the muscular and the nervous. If this remarkable autopsy does not admit of our recognising whether the lesion began in the nerve or the muscle, it authorizes us, at least, to affirm that the lesions were parallel and identical in both these tissues.

When I spoke of the pathological anatomy of progressive muscular atrophy, I reminded you that the absence of all kind of lesion of the nerve-centres was in accordance with the absence of nervous disorders during life. Scarcely is there, in some cases, cutaneous anæsthesia of the regions corresponding

to the degenerated muscles, and this is the only nervous symptom ever observed. Everywhere else the integuments retain their sensibility, free from exaltation or diminution. The organs of the senses are in nowise disturbed in their functions. Lastly, intelligence remains entire unto the end; and it is as curious as it is sad to see poor unfortunates—who are, as it were, reduced to the condition of skeletons, through the loss of a great portion of their muscles—not only perform all the functions of organic life, but possess a perfectly lucid mind, and thus witness themselves their slow and progressive destruction.

I remember, gentlemen, the history of a lady at Tours, whom the illustrious Bretonneau attended for several years, and who, according to the description given me by my old teacher, died in the last stage of muscular atrophy. She could scarcely breathe, and no longer spoke; but her eyes still retained all their vivacity, and reflected her intelligence, which did not forsake her. She could still contract some of the muscles which support the head, and those of the index-finger of her right hand. During the last days of her existence, she conversed with her children by means of this finger. Several sets of alphabetical letters, like counters, had been got for her, and with her finger she put the letters together, composing words and sentences. By that means she was enabled to make her will.

The epithet *progressive*, applied to the word *atrophy*, sufficiently indicates the course of the disease. If it be not rare to see it remain stationary, after having affected one or more regions, too often, after a more or less prolonged pause, it spreads to other parts; and as I have already told you of the manner in which it progresses, in the rapid sketch which I gave you of the symptoms of the disease, I need not revert to it now. If we only look at what occurs in one or several muscles taken singly, the course of the disease is rapid, for the tissues may be completely destroyed in a few months; but if we look at the disease as a whole, and calculate the time which has elapsed from the period when the first symptoms showed themselves until the fatal termination, the duration of progressive muscular atrophy is generally long. If in some cases, as Dr. Duchenne (de Boulogne) has had occasion to observe, in less than two years a good many muscles of the upper limbs and the trunk, some of those of the lower extremities, the muscles of the face, those of respiration and deglutition, have been transformed in various degrees, the disease usually progresses much more slowly, although its duration is never very determinate.

On this point, therefore, we have no precise data for prognosis. But we know, unfortunately too well, that the prognosis is invariably most serious. Death, indeed, may be the consequence

of these grave disorders of the locomotor apparatus. It may be directly brought on when the atrophy invades the muscles of deglutition and respiration, and the patient, as I told you, ultimately dies of starvation or by asphyxia; or it may be caused indirectly when, as I have already told you, there supervenes an intercurrent affection, bronchitis for instance, to the evolution of which the muscular atrophy adds a fatal complication. But even supposing that this fatal termination occurs as late as possible, and that, from the disease remaining localized, the patient suffers for many long years, the prognosis is not the less unfavourable on this account. The disease may, indeed, pause in its course, it may be arrested in its progress, but we cannot hope to see it retrograde, for the muscles that have been destroyed are ruined for ever. You may imagine, gentlemen, the infirm condition to which the unfortunate patient, thus deprived of a greater or less portion of his muscular system, is condemned —a condition which is all the more cruel, that progressive muscular atrophy much more frequently attacks individuals in the strength of years, or belonging to the working-classes, and who need all the freedom of their movements in order to earn their living and that of their family.

With regard to prognosis, there is another peculiarity mentioned by Dr. Duchenne (de Boulogne), and which to me also appears of great importance—namely, that the disease becomes general, and terminates fatally, all the more quickly that no appreciable determining cause has brought it on. It then seems that the diathesis, of which the muscular lesion is only an expression, is much more active than when the disease has been excited by a determining cause. Lastly (and this is also a remark made by Dr. Duchenne), when the disease primarily attacks the muscles of the trunk, it remains stationary much longer, and spreads to other regions more slowly, than when it sets in first in the muscles of the limbs.

A disease which has such a characteristic physiognomy cannot be mistaken by a careful physician who has once seen it. There are cases, however, in which the *diagnosis* is attended with some difficulty. When, for instance, a considerable degree of plumpness prevents the production of the characteristic deformities, the disease may at first sight be unobserved, although from the disorders of locomotion it can be easily recognised.

Rheumatic pains are sometimes followed by muscular atrophy, which it is important not to confound with progressive atrophy, for the two affections are essentially different. The course and duration of the symptoms, the forms which they assume, and the seat which they select, suffice, before having recourse to the test of electricity, to guard one from error. Rheumatic atrophy is,

indeed, preceded by more or less violent pains, which are intensified by the voluntary contraction of the affected muscles, and by pressure made on them, whilst progressive atrophy is generally painless. Besides, the whole muscular mass is involved, and not a few bundles only, as in progressive atrophy. Lastly, whilst in the latter electric contractility is considerably weakened in proportion to the number of muscular fibres destroyed; in rheumatic atrophy, on the contrary, whatever be its stage, and however restrained and impossible even voluntary movements may have become, galvanization is still all-powerful, for the muscle has merely diminished in size, and its fibres have undergone no transformation.[1]

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[1] [Muscular atrophy has been known to follow prolonged neuralgia in a limb, through a reflex action; and Notta (in "Archives générales de Médecine" for November 1854, p. 557) relates seven instances in which this occurred. Dr. Brown-Séquard (in his "Lectures on the Central Nervous System," p. 163), quotes a case of Dr. Clément Bonnefin, in which the atrophy was clearly traceable to neuralgia, and mentions two others which came under his own observation. In one of these, wasting of some of the muscles of the leg was brought on by sciatica; in the other, pain, starting from the cicatrix of a wound on the *left* forearm, produced atrophy of *both* arms.

I have myself had under my care, at the National Hospital for the Paralysed and Epileptic, a woman affected with marked atrophy of the muscles of the right lower limb, consequent on prolonged and severe sciatica.

These cases may be at once distinguished from real progressive muscular atrophy by the galvanic test, the wasted muscles retaining unto the end their electro-muscular contractility.

There is a peculiar affection, spoken of by French writers as *marasme essentiel*, which may be easily mistaken for progressive muscular atrophy. Indeed, it essentially consists in gradual wasting of the muscles, and as such, after all, is entitled to the denomination of muscular atrophy; but it is atrophy which differs *in toto* from that which occurs in the form of disease described by Professor Cruveilhier, now generally known as progressive muscular atrophy. In that rare and peculiar affection, of which I have within the last twelvemonth seen two well-marked instances, the wasting is general, and affects all the muscles, and all the bundles of the same muscle, equally and uniformly. The patient gets gradually thinner and thinner, and assumes after a while the aspect of a living skeleton. He complains of a great sense of cold, particularly in the extremities; and he is generally a hypochondriac, and often presents the characters of that exaggerated form of hypochondriasis, which has been so fully investigated by Dr. Bouchut, of Paris, and called by him *nervosisme chronique*. One of the cases, which have come under my own notice, occurred in the private practice of my friend and colleague Dr. Ramskill, at whose request, when the patient died, I made a post-mortem examination. The wasting of the whole muscular apparatus was peculiarly striking; but the muscles, however diminished in bulk, still retained their normal red colour, while under the microscope they exhibited their transverse striæ unaltered and unchanged. The other case was that of a gentleman between 35 and 40 years of age, whom I saw in consultation with my friend Dr. Brown-Séquard. He was of very tall stature, and his extreme emaciation made him look still taller. At first sight he looked the subject of general progressive muscular atrophy; but the uniform manner in which the muscles were involved, including those of the face, pointed to the real nature of the case, while all uncertainty was set at rest by the electric test, every one of his muscles responding quickly and normally to a galvanic

The differential diagnosis between lead-palsy and progressive muscular atrophy is attended with greater difficulty. In the former of these affections, however, electro-muscular contractility is completely abolished, whereas in the latter it is only diminished in proportion to the number of muscular bundles which have undergone the characteristic alteration. Moreover, even when saturnine paralysis has become general, the electric contractility of certain muscles alone is destroyed—namely, of the extensors of the hand on the forearm, and subsequently of the muscles of the arm, chiefly the biceps and deltoid. Lastly, the evolution of the symptoms (for in the lead-disease palsy precedes atrophy), the previous history of the patient showing that he has been exposed to the influence of lead, and other morbid phenomena special to saturnine poisoning, will throw light on the question.

The disease which most closely resembles progressive muscular atrophy is the atrophic paralysis of infants, but the latter differs from the former in being complicated by an arrest of development of the bones of the limb whose muscles have undergone fatty degeneration.

It is important also to distinguish from progressive atrophy, the wasting which is due to an injury to a nerve or the branch of a nerve. The wasting is, in such cases, exclusively limited to the groups of muscles supplied by the nerve or the branch of a nerve, and the complete limitation of the disease must immediately suggest the idea of an equally circumscribed lesion, and do away with that of progressive muscular atrophy, in which the disease settles here and there, at random as it were, and not according to the anatomical distribution of a nerve. There is even now, in the St. Jane Ward in this hospital, a man suffering from wasting of the left hand and forearm, and whose tongue is also atrophied in its left half. But it is easily recognised that the wasting involves the deep flexor of the fingers, the interossei, the two inner lumbricales, and the deep adductor of the thumb—that is to say, muscles supplied by the ulnar nerve. Now this man has had syphilis, and has now an exostosis on a level with the epitrochlea, which, by compressing the ulnar nerve, has probably altered its structure, and then caused wasting of the muscles which it supplies. It is pretty probable that another exostosis, depending on the same cause, compresses either the lingual nerve or the hypoglossal, and causes, in the

excitation. Duchenne (*loc. cit.* p. 529) relates a very interesting case of this kind, which was under Dr. Vigla's care in one of the Paris hospitals; and mentions two other instances which he saw himself—one at the Lariboisière, and the other at the Charité Hospital. Such cases may be immediately recognised from Cruveilhier's atrophy by the persistence of electro-muscular contractility, and the uniform and equable manner in which all the muscles are affected.—Ed.]

muscles of the tongue, alterations of nutrition similar to those of the muscles of the arm.

Lastly, there is a disease of which few instances will come under your notice, which also causes muscular atrophy—namely, the *dry leprosy* of hot climates. But in this case also, the wasting is circumscribed, and limited to all the muscles of the hand. The skin of the hand is red, thickened, and completely anæsthetic; the fleshy masses have entirely disappeared, and the fingers assume the shape of claws. Such were the symptoms noted in a woman whose case M. Peter had occasion to study for a long time, whilst he was Dr. Cruveilhier's resident assistant. The differential diagnosis is easy, for apart from the leprosy, which is easily recognised, and from the complete anæsthesia which accompanies it, the flexor muscles of the fingers are found to be retracted, a fact on which Dr. Duchenne justly lays particular stress.[1]

I told you, a moment ago, that progressive muscular atrophy never terminated in death so rapidly as when it was developed without any appreciable determining cause. The most frequent of such causes is continuous and excessive work, necessitating the exercise and exaggerated contraction of certain muscles. But determining causes are subordinate to an individual predisposition, to a diathesis which is more generally acquired, but is often also transmitted from one generation to another.

This influence of hereditary predisposition, or at the least of consanguinity, was, in 1851, pointed out by Dr. Meryon, who, in a communication to the Medico-Chirurgical Society of London, related the history of a family, in which three boys suffered from muscular atrophy. I have already mentioned to you similar instances, observed by Dr. Duchenne. The most remarkable point in Dr. Meryon's case is, that of the nine children composing the family, the three sons were alone affected, whilst the six daughters escaped.[2]

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[1] [The wasting of the muscles of the hand in the dry form of leprosy may probably be accounted for by the presence of a neuroma, which, according to the observation of some medical friends of mine in practice in the island of Mauritius, is in all such cases developed in the substance of the ulnar nerve, a little above the inner condyle of the humerus.—Ed.]

[2] [See "Medico-Chirurgical Transactions," vol. xxv.—The family consisted of four sons and seven daughters. At the time of the above communication, only three of the boys had been affected with the disease, but in his work on Paralysis (at p. 205), Dr. Meryon states that "three sons have died of the disease, and the fourth is now the subject of it; the seven daughters are alive and perfectly healthy."

The same author cites, on the authority of an eminent physician, the case of a very stout-made, well-formed, and in every respect healthy boy, who at the age of 10 began to show weakness in the lower extremities, which gradually increased, and eventually required him to ask for assistance to raise himself from the floor. The hereditary transmission of the disease was very distinct in

It is, besides, an observed fact that this disease rarely attacks women, and that in them it does not become general, no instance of its doing so having been recorded as yet.[1] When I have added that muscular atrophy is scarcely ever met with but in adults, I shall have told you the little that is known concerning its etiology.

Treatment is, unfortunately, powerless against this complaint; and if localized faradization has, in some cases, been able to arrest its development, the disease has made further progress, after a more or less prolonged intermission.[2]

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this case. His mother had three married sisters: one, who very early became a widow, has one daughter, now grown up and quite healthy. Another had two sons and one daughter. Both of the sons, at a very early age, became affected in a manner similar to that described above. The loss of power gradually extended to the lower extremities till they became quite helpless, requiring to be lifted from one chair to another, and wheeled about from place to place.

The third sister had a family of two daughters and one son, a very fine boy. He also began very early to show some peculiarities in his gait, which gradually increased; and when he was 10 years of age, there was no apparent prospect of improvement. The two daughters were perfectly healthy. ("On the Various Forms of Paralysis," by Edward Meryon, M.D., p. 206.)

In a family known to Oppenheimer (quoted by Dr. Roberts, in his essay on Wasting Palsy, p. 137), "two uncles and a cousin were already deceased, while another cousin (both cousins were males) and two brothers still suffered from wasting palsy. Another had lost his father by the same complaint."

In none of the cases which have come under my own observation, have I been able to trace any hereditary predisposition to the complaint. Exposure to cold and wet, combined with poor living and consequent malnutrition, seems to have been the probable cause at work in the production of the disease. With regard to *excessive muscular exercise* as a cause of atrophy, as pointed out first by Darwall, and endorsed by Aran, Wachsmuth, Roberts, and Duchenne (de Boulogne), no very conclusive evidence has been adduced in support of this view. In no case yet have I been able to ascribe the disease to this cause; and Aran himself was obliged to acknowledge that there was an immense number of persons pursuing the same occupations as his patients, and with the same ardour, in whom nothing similar was observed, although he is right in adding, that "if there be a predisposition, those members which are the most fatigued will certainly be the first assailed."

If unduly prolonged contraction be the cause of progressive muscular atrophy, it is difficult to understand why an entire muscle does not waste away, instead of individual bundles only, and why all the muscles of a limb which concur in the production of a combined movement do not atrophy simultaneously and to an equal degree; why, for instance, the disease (as it sometimes does) jumps from a flexor to an extensor muscle, or *vice versâ*. On the other hand, the influence of cold in originating this curious disease, can be better understood from its constricting action on the capillary vessels, and the consequent diminution of the amount of blood distributed to the part. If this influence be kept up for a prolonged period, or repeated so often as to border almost on a continuity of action, the circulation of the part may be so materially interfered with as to deeply modify its nutrition, and thus destroy the healthy balance between repair and waste.—Ed.]

[1] [I have at present under my care a woman who is affected with general progressive muscular atrophy, and the disease is so far advanced that there is wasting of the intercostal muscles and complete atrophy of the tongue, with difficulty of deglutition and respiration.—Ed.]

[2] Dr. Duchenne (de Boulogne) speaks very hopefully of the influence of

Before dismissing the subject of progressive muscular atrophy, I wish, gentlemen, to discuss with you an important question,—namely, whether this singular disease is an affection of the spinal cord, or whether it should be grouped with diseases of the muscular system. Those who ascribe the atrophy to a spinal lesion ground their opinion on the results of post-mortem examinations, which have shown a singular diminution in size of the anterior roots of the spinal nerves. This pathological change is perfectly evident in the specimens which I now show you. M. Sappey has had the extreme kindness to prepare, with the greatest care, the spinal cord of my patient, and has placed by the side of it several cords taken from the bodies of individuals who had not suffered from any nervous disorder. You can see how great the difference is between them. It is supposed, then, that this lesion of the anterior roots is the starting-point of the functional disorders, and that the muscles, no longer receiving any nerve-force as usual, first atrophy, and next become altered in their structure. They atrophy in the same manner as muscles do in cases of saturnine or rheumatic paralysis.

This view of progressive muscular atrophy is at first sight seductive, but it cannot be accepted when the question is closely examined.

First of all, the anatomical fact, namely, the atrophy of the

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faradization, not only in arresting the disease, but also in restoring, to a certain extent, the nutrition of the affected muscles, provided, however, their tissue should not be already altered. So long as the atrophy is confined to a diminution in the size or bulk of a muscle, and its ultimate fibres are not disintegrated, he thinks that electricity can rouse the vitality of the muscle, and even give rise to the production of new fibres. In order to obtain this beneficial result, the muscle must respond to electric excitation, for all hopes must be given up when electro-muscular contractility is entirely and completely abolished. The rules which he lays down are, that a very powerful instrument be used, with very rapid intermissions; the intensity of the current is to be diminished, in proportion as muscular sensibility returns; and it is of the highest importance that the patient is galvanized for five or ten minutes only, as otherwise the disease will be aggravated, and the remaining muscular fibres will waste away more rapidly.

Dr. Meryon speaks very favourably of the influence of arsenic in arresting the disease, while Dr. Heltzlar (of Aix-la-Chapelle) goes so far as to say that he has cured several cases of progressive muscular atrophy by the administration of sulphur-baths. I have repeatedly tried arsenic, but cannot say that I have been able to obtain any satisfactory results from it. On the other hand, I have seen a general tonic plan of treatment, the administration of preparations of iron and of cod-liver oil, together with localized faradization, in some instances arrest the disease for a while, by improving the general nutrition of the body. As the nature of the wasting is different from that of fatty degeneration, there is no reason why cod-liver oil should not be given with a view of improving nutrition. The excellent effects of the oil in rickets are well known; and if there be some analogy, as I believe with Dr. Meryon, between this affection and Cruveilhier's disease, a remedy which has been found so beneficial in the one is likely to be useful in the other.—Ed.]

anterior roots, proves nothing as to the priority of the nerve-lesion. It is known that if the brachial plexus and the nerve-roots which concur in its formation be dissected in an individual whose arm has been amputated, the nerves are found to be atrophied as far as their entrance into the fissure of the spinal cord. In individuals who have lost an eye, not from cerebral disease, but in consequence of ophthalmia or of a wound, the optic nerve is invariably found to be atrophied as far as the commissure. The peripheral lesion and the cessation of function may, therefore, be the cause of the atrophy of the nerves; and it may be reasonably maintained that the atrophy of the anterior roots, found after death, in nowise decides the question at issue. But I pass on to other arguments. If the starting-point of progressive muscular atrophy were in the spinal cord, we should always see the disease affect a whole group of muscles, and not a few only.

Thus, for example, all the muscles which derive their nerves from the brachial plexus would be involved simultaneously and to the same degree; or, at the very least, all the muscles supplied by nerves from one of the cords of this plexus would atrophy and degenerate at the same time. But such is not the case. In one patient we see a perfectly isolated muscle, in the hand for example (and this is the most frequent case), atrophy completely and lose its structure and functions, whilst other muscles, supplied by the same nerve-branch, are untouched. We see at the same time one or more muscles of the arm, trunk, and lower limbs, on either side indifferently, becoming affected in turn—a circumstance which is never observed in those diseases in which the primary lesion of the nerve-centres and the nerve-cords cannot be called in question.

I confess, gentlemen, that a careful study of the course of this disease does not allow me to group it with diseases of the spinal cord, in spite of the autopsies made by a celebrated anatomist, Professor Virchow, who declares that he has seen, with the aid of the microscope, alterations in the anterior columns of the cord itself. I dare not, on the other hand, adopt a perfectly opposite opinion, and say that progressive muscular atrophy is primarily a disease of the muscles. It is infinitely probable that the nerves of organic life which accompany the terminal and muscular branches of the arterial tree, are so modified in their functions as deeply to disturb the nutritive phenomena over which they preside; but when we find grave organic lesions and degenerations of tissue in a kidney or in the liver, although in every case, perhaps, the vaso-motor nerves may be at fault, we do not the less say that the lesion is renal or hepatic, if the supposed nervous disorder exist only in the kidney or liver. Until more is known on the subject, we may justly regard progressive

muscular atrophy as a disease of the muscular system, if we wish to conform ourselves to the usual view taken of anatomical lesions; although this does not prevent us from admitting the existence of a diathesis which influences the whole system, and predisposes it to the strange lesions which we have just studied.

APPENDIX BY THE EDITOR.

[It is an open question still, whether progressive muscular atrophy is an idiopathic affection of the muscles themselves, as rickets is of bones, or whether it is dependent on structural alteration of the spinal cord. Dr. Meryon, who holds the former opinion, has supported it by the results of two carefully-made microscopical examinations of the cord, in undoubted instances of the disease, and even cites a case in which the bones seemed to participate in the inadequate nourishment of the muscles. "The subject of this case was a boy, of 8 years old, who twice broke his thigh by simply falling on the floor—once when putting on his coat, and the second time when attempting to walk across a room."

In many instances of progressive muscular atrophy in which the cord was examined, no trace of disease was found. Of the 105 cases collected by Dr. Roberts, in his valuable essay on Wasting Palsy, the cord was examined in 13, and found altered in 4 only.

On the other hand, instances have been recorded in which the anterior roots of the spinal cord, and even the anterior columns themselves, were found reduced in size; and it has been therefore assumed by some, that progressive muscular atrophy is due to disease, to wasting of those parts of the spinal cord. But those changes are by no means constant, for they have not been met with, although looked for by competent observers. Thus Dr. Duchenne (de Boulogne) relates the case of a man, named Lejeune, who was an in-patient at the Charité Hospital, under Dr. Andral's care, and in whom no lesion of the anterior roots of the spinal nerves could be detected. The same author cites also a case, observed by Oppenheimer and Hassé, in which a microscopical examination of the *nerve-centres*, of the cerebral and spinal origins of the nerves, and of their peripheral trunks, disclosed no alteration whatever; and in three patients, who died in the Heidelberg Hospital, under Dr. Friedreich's care,

the anterior roots of the spinal nerves were found normal, when examined under the microscope.

Within the last few years, however, some admirable microscopical examinations of the spinal cord, made in this country by Mr. Lockhart Clarke in cases of muscular atrophy, have tended to shake the belief, which seemed to be gaining ground, that the cord itself was not primarily affected in Cruveilhier's disease. The elaborate care with which these examinations were made leaves nothing to be desired, and the intimate acquaintance which Mr. Lockhart Clarke has with the normal structure of the cord renders him peculiarly competent to detect any, however trifling and minute, departure from healthy organization of that nerve-centre. The ingenious manner in which he prepares nervous tissues for microscopical examination, seems the best adapted for bringing out every change of structure in them.

In three cases, regarded as instances of progressive muscular atrophy, Mr. Lockhart Clarke has examined the cord, and found in the *grey substance* in those regions of the cord which supplied the wasted muscles, and generally around or in the vicinity of bloodvessels, numerous patches of transparent granular degeneration. The nerve-cells also contained an unusual number of coarse pigment-granules, while corporea amylacea were abundantly deposited around the central canal. To these three cases may be added a fourth, that of a patient who died of typhus-fever in Guy's Hospital, under Dr. Gull's care, and which is published in the eighth volume of "Guy's Hospital Reports" (1862). In this case "the hands alone were affected, the patient complained of no pain, but only of weakness and a feeling of numbness. He could move both thumbs and index-fingers freely; he could also extend the first phalanges of the other fingers of both hands, but not in the least degree the second and third phalanges, which were gently flexed towards the palm. The interosseous spaces on the backs of the hands were sunken from the wasting of the muscles. The palms of the hands were hollow, and the flexor tendons very prominent. The thenar eminences were wasted, and the hypothenar almost entirely gone, particularly on the right side. The motion of the wrist-joints was unaffected. He could move the arms freely in all directions, and he could walk perfectly well." On the patient dying of typhus-fever, his spinal cord was examined. "The exterior presented nothing, except that the cervical enlargement appeared broader and somewhat flattened. On making transverse sections, the white columns had their normal consistence and texture, but the centre of the cord had a large cavity—beginning at the fifth cervical, enlarging to the seventh, and

from thence tapering downwards. The only remains of the grey matter were at the anterior part of the cavity behind the anterior columns. Here the caudate vesicles had their normal size and structure; the pigment, nucleus, and nucleolus being well marked, and the tubular structure unaltered. The cavity in the cord was bounded by a layer of condensed grey substance, which could be separated as a distinct membrane. On its interior surface, forming the lining of the cavity, were a number of delicate, elongated nuclear bodies, apparently epithelium. The cavity contained a clear fluid. One or two granular cells were found scattered among the white columns, but no further traces of any active tissue change. The roots of the nerves appeared normal, and contained healthy tubules."

This case is certainly one of progressive muscular atrophy in an incipient stage; and it is of considerable importance, as showing the coexistence of a central nerve-lesion with characteristic wasting of some of the muscles of the hands. But still some doubt must remain in one's mind as to the exact relation between the two sets of lesions, the spinal and the muscular, and as to the former being the true cause of the latter. The only muscles affected were those of the hands, and a few of them only. Now, the post-mortem examination disclosed the existence of a large cavity in the centre of the cord, "beginning at the fifth cervical, enlarging to the seventh, and from thence tapering downwards." The lower limit of this cavity is not specified, but from the woodcut which is given with the report of the case, the cavity may be seen to extend a good way down the cord. How then, it may be asked, can one account for the restricted localization of the atrophy in some of the muscles of the hands, if this atrophy were due to the absence or destruction of the central grey matter of the cord? If this were the cause, why was not the effect commensurate with it? A lesion of very limited extent in the nervous centres is often followed by morbid manifestations over large areas of the body; but extensive disorganization of these centres is not, as a rule, accompanied by symptoms of disease in excessively limited parts of the body. The nature of the lesion found in the spinal cord, in this case, is so essentially different from the alterations of structure met with in other instances, that it may well be asked whether this absence of the central grey matter of the cord was not a *congenital defect*, and whether it had anything to do with the atrophy of some of the muscles of the hands?

Of the three cases in which Mr. Lockhart Clarke examined the spinal cord, one alone seems to have been a clear and undoubted case of progressive muscular atrophy. The patient, Dr. P——, was seen during life by several eminent physicians, and the case is related at full length by Dr. Gairdner, in the

third volume of Beale's "Archives of Medicine." There was one symptom, however, in this case which is not, as a rule, met with in progressive muscular atrophy—namely, *acute pain*, which preceded and accompanied the wasting, and was also complained of in parts (such as the feet) that were not atrophied. In those very rare instances of Cruveilhier's disease in which pain has existed, it has only been complained of in parts affected with wasting, but never all over the body. Thus, in a case recorded by Sir Charles Bell,[1] it is stated that "the affection had commenced three years previously, with a pain extending from the left shoulder to the elbow, which the patient was obliged at the same time forcibly to extend. The emaciation of the muscles of the thumb was accompanied by a constant and violent pain."

In Dr. P——'s case, agonizing pain was complained of all over the body, and the patient, throughout his illness, often spoke of himself as suffering "torture." These symptoms may well be accounted for by the irritation of the sensitive nerve-fibres passing through the central grey matter of the cord, consequent on the changes going on around the central canal, and attended with the deposit of corpora amylacea. For it is stated that "changes of structure were found in the posterior grey substance, extending in a variable degree from the lower end of the cervical enlargement to the third cervical nerves, and a considerable deposit of corpora amylacea around the central canal, chiefly in the cervical, and less in the dorsal and lumbar portions of the cord."

It is not clear, however, how it happened, that with such extensive changes as those found after death, even in the *dorsal* and *lumbar* regions, the atrophy did not affect the muscles of the trunk and those of the lower extremities. However important and valuable this case may be, therefore, it does not yet set the question at rest, whether progressive muscular atrophy is due or not to alterations of structure in the grey matter of the cord.

The other two cases, in which Mr. Lockhart Clarke made a most careful microscopical examination of the spinal cord, do not appear to be genuine instances of Cruveilhier's disease. In fact, one of them is published by Dr. Radcliffe (under whose care the patient was in the Westminster Hospital), under the heading of "Paralysis with Muscular Atrophy."[2] The history of the case; the course and progress of the disease, as far as they could be ascertained; its first manifestation a month after a sunstroke in California, followed two months afterwards by an assault by

[1] "The Nervous System," 3rd ed. London, p. 432.
[2] Medico-Chirurgical Review, vol. xxx. (1862).

ruffians, who left the patient stunned and insensible, do not point to an uncomplicated instance of progressive muscular atrophy. As to the symptoms during life, they were amply accounted for by the presence of extensive changes of structure, found not in the spinal cord alone, but in the medulla oblongata also. "Not only was the grey substance found diseased, but all the white columns of the cord in every region, but particularly in the cervical region, had suffered from atrophy or degeneration. In the cervical region, the anterior roots of the nerves were decidedly below their average size. In the nerve-fibres, the change was observed chiefly in the axis-cylinders, which were frequently reduced to less than one-half their normal diameters. . . . The medulla oblongata was also diseased, and the morbid changes were very extensive and very marked, from about the lower end of the olivary bodies to the commencement of the fourth ventricle—*i.e.*, over a space including the principal part of the grey tracts or vesicular centres, from which the hypoglossal nerves take their origin. (The tongue was considerably wasted in this case, and the patient could not speak.) The roots of these nerves, in their course through the medulla, were in some places not more than half their natural size, and in other places could scarcely be discerned."

In Mr. Lockhart Clarke's third case, extensive disorganization of the spinal cord, in nearly all its length, was found after death. The patient was under Dr. Thudichum's care, and the case is published in Beale's "Archives of Medicine" (No. 12, vol. iv.) It does not, however, present the characters of true progressive muscular atrophy, or Cruveilhier's disease. Paralysis was the first symptom to show itself, and wasting only made its appearance subsequently. True, it set in very rapidly, and could not be accounted for by inactivity of the muscle, by disuse; but it was, nevertheless, unmistakably preceded by paralysis, a fact directly at variance with the natural history of Cruveilhier's disease, in which wasting is the primary and the prominent symptom, and paralysis only comes on gradually, and as an ultimate consequence of the destruction of the muscular fibres. Another circumstance in this case which makes it differ from true progressive muscular atrophy is, that all the muscles of the limbs were affected *en masse* and at once; whereas in Cruveilhier's disease, the rule is that the muscles are involved separately and successively, and individual bundles of the same muscle even. Again, microscopical examination of the wasted muscles did not, in Dr. Thudichum's case, disclose exactly the same alterations of structure as those which characterize Cruveilhier's disease. Thus, it is stated that "the paralysed muscles remained of a pale rosy hue when exposed to the air, while the muscles which had been contractile became

dark-red as usual. Examined with the microscope, the paralysed muscle exhibited the fibrils in a state of relaxation. While in the healthy muscle the fibrils are closely folded together, so as to produce the transverse striæ, in the paralysed muscle all transverse striæ had disappeared, and the fibrils lay in undulating lines. Beyond that there was no change even in the most wasted muscle. There was no fatty or other deposit of any kind, either in or upon the microscopical elements." How different these appearances are from the changes in structure which characterize true progressive muscular atrophy—the pale-fawn colour of the wasted muscles, the complete disappearance not only of the transverse but, after a time, of the longitudinal striæ also, the collapse of the sarcolemma and occasional disruption of this sheath, and, lastly, the breaking-up of the muscular fibre into granules—need be merely pointed out.

However valuable and interesting the results arrived at by Mr. Lockhart Clarke may be, they yet cannot be said, when his cases are critically examined, to have settled the question at issue, whether progressive muscular atrophy is really due to disease of the grey matter of the spinal cord? But it must be added, that it will be only by examining the spinal cord in cases of Cruveilhier's disease with the same elaborate care and in the same admirable manner as Mr. Lockhart Clarke, that this important point can ever be settled. Nor will this end be attained until the deplorable confusion which still exists between cases of true progressive muscular atrophy and of spinal paralysis with rapid wasting of the muscles be done away with. Microscopical examination of the wasted muscles should always precede, and be regarded as important as, that of the nervous centres; and, if necessary, the condition of the muscular tissue may be determined during the patient's life, by examining under the microscope a piece of the affected muscle, picked out either by means of a hook, like the one used by Dr. Thudichum in cases of trichiniasis, or by a still more ingenious instrument, which Dr. Duchenne (de Boulogne) frequently uses in cases of paralysis of old standing, in order to determine the condition of the muscles.

Progressive muscular atrophy has been suspected by some to depend on disease of the sympathetic, and in one case Professor Schneerogt, of the Hague, found an alteration of the splanchnic nerves.[1] But it is not probable that this opinion is correct, for there is, in this complaint, a total absence of all visceral disorders, and this is a fact not easily reconcilable with an affection of the sympathetic. It is a remarkable circumstance also, that pale unstriated muscular fibres escape in progressive

[1] See Canstatt's "Jahresbericht," 1855.

muscular atrophy, and that the heart, the walls of which consist of striated fibres, is not affected. In a case, however, mentioned by Dr. Fuller (in the course of a discussion on progressive muscular atrophy at the Medico-Chirurgical Society of London) as having been seen by himself and Dr. C. J. B. Williams, the heart, about two months before the patient's death, became extremely feeble in its action, and the pulse fell to 26 in the minute.—Ed.]

LECTURE IX.

FACIAL PARALYSIS, OR BELL'S PARALYSIS.

Facial Hemiplegia: its causes and symptoms.—Contraction of the Muscles consecutive to Paralysis of one side of the Face may be mistaken for Paralysis of the opposite side.—Treatment.—*Double Facial Paralysis.*

Gentlemen,—Facial paralysis is an affection which is often met with in practice, and although it is in general of no gravity, yet treatment, unfortunately, fails too often to cure it. However mild the disease may be in the majority of cases, it sometimes excites singular alarm in the patients and those around them; and it is all the more important that the physician should know well how to recognise it, that it still pretty frequently gives rise to lamentable errors of diagnosis. In order to put you on your guard against such mistakes, I wish, in this conference, to call your attention to some special points relating to this subject, *à propos* of two individuals suffering from this paralysis whom you have seen—one in St. Agnes Ward, and the other in the neighbouring ward of St. Louis.

The young man in St. Louis Ward is 17 years old. He tells us that, through his being prevented from working by a slight wound in his hand, he spent his time in the streets and in public promenades; that on Monday last he slept in the open air on a heap of pebbles; that he was in a state of perspiration at the time, and got cold after falling asleep. He went home in the evening, feeling uncomfortable. The next morning, however, he got up as usual, feeling absolutely no disturbance of his health; but when he began to eat, he felt something peculiar, and had some difficulty in masticating. When his food got between his right cheek and his teeth, he was compelled to squeeze the cheek with his hand, so as to push the food between his teeth again. He was surprised at this, and could not account for it, as it was unaccompanied by any painful sensation. He felt more surprised when one of his friends, on seeing him, told him that his mouth was awry, and that it became considerably more so whenever he laughed. On then looking at himself in a glass, he verified the fact, and feeling frightened, came to the hospital to be cured.

The following points I ascertained myself: When the patient's face is at rest, the right side merely looks slightly flatter, and

more flaccid than the left; his right eye is also more widely open than the left, but his physiognomy, after all, does not look strange. When he speaks, and still more when he laughs, the left angle of his mouth is immediately drawn upwards and outwards, while the right one is perfectly motionless. As the eyelids, the cheek, and the lips are motionless also, the face has in consequence a singular expression, especially when the patient tries to contract his muscles. The eyelids being motionless on the right side, the right eye cannot be completely closed; but the globe of the eye itself moves perfectly, at the patient's will, to the right or to the left, upwards or downwards. Sight is in nowise altered. The motor muscles of the eye are not therefore in the least at fault, and the paralysis, (for there is paralysis present) affects exclusively the orbicularis palpebrarum, without involving the levator palpebræ superioris.

When the patient is asked to put out his tongue, he does so with perfect regularity; and the difficulty which he has in articulating certain words is not owing to defective action of the muscles of that organ, but to the immobility of the right cheek. On examining the fauces, it is evident that the double arch formed by the pillars of the soft palate and the mouth has not on both sides the regular form which it normally has, for the left arch is narrower than the right, showing that the uvula inclines to the left.

I have told you how the complaint originated. Save a few hours of malaise, the patient has never felt any general disturbance, or the slightest headache; nay, more, he states that he has never felt better, and that his appetite is twice as good as formerly. I do not, of course, attach great importance to what he says, because he is doubtless prompted by the fear of being put on too strict a diet. This circumstance is sufficient, however, to show that there has never been any disturbance of his health, and that his complaint consists merely in a motor affection of the muscles of the face, the cutaneous sensibility of which is in nowise perverted. As to other locomotor apparatus (the limbs, for example), their functions are discharged perfectly. The case, then, is one of that form of paralysis which has been named *Bell's paralysis*.

In the case of the other man, who occupies one of the first beds in St. Agnes Ward, the paralysis of the face occurred under different circumstances. His health has been generally good, and he was smoking his pipe at a window during the heavy storm which burst over Paris a few days ago. A sudden and violent thunderclap in the neighbourhood frightened him very much, but, laughing at his terror, he soon resumed his place at the window, and went on smoking; but he perceived that he

had some difficulty in spitting out, and a few moments afterwards, his wife noticed that his face was distorted. As a few days elapsed without this distortion disappearing, he felt anxious about it, and came to the Hôtel-Dieu. In this case, then, mental emotion, intense fright, brought on the same complaint as cold did in the case of the young man in St. Louis Ward. In both these men paralysis of one side of the face set in, impairing movement alone, and involving exclusively the muscles supplied by one of the seventh pair of nerves. These two cases are instances of that kind of facial hemiplegia which has been termed idiopathic, in the language of schools; meaning thereby that the complaint occurs independently of all appreciable material, traumatic lesion, whether inflammatory or not, affecting the facial nerve primarily or secondarily.

I shall now rapidly review the different *causes* under the influence of which facial paralysis may occur.

Cold is one of the most frequent of these, and it would not be difficult to collect a great number of cases analogous to our first one, for this kind of paralysis has for a long time been spoken of by authors under the name of *rheumatic paralysis*. The patient is seized in the midst of the most perfect health, without there being any disturbance of the general economy: a mere draught, residence in a damp place, or in a newly-built house, may bring it on.

You have seen that *mental emotion* may cause it, as in the case of the patient in St. Agnes Ward who was greatly frightened. In others the paralysis came on after a violent fit of anger, and in others, again, after some profound grief, caused, for instance, by the unexpected death of a dear friend. Sometimes, also, the disease cannot be ascribed to any appreciable cause.

In all the cases in which it is not owing to the presence of an appreciable material lesion, the disease sets in suddenly; and the same thing happens when the paralysis results from *traumatic lesions* of the nerve.

You know, gentlemen, that it is not of uncommon occurrence to meet with facial paralysis in newly-born children, and that it is sometimes mistaken by careless persons for a symptom of cerebral disease. This paralysis, which is due to the compression by the forceps of the facial nerve as it emerges from the aqueductus fallopii, is generally transitory and of no gravity whatever; when the compression, however, has been excessive, it may persist through life.

Your professors of surgery have pointed out to you this traumatic cause of facial hemiplegia, and they have also taught you that this paralysis could be the consequence of wounds of the seventh pair, inflicted either by accident, or during a surgical operation.

This form of paralysis may result also from a fracture of the skull, involving that part of the temporal bone in which lies the aqueductus fallopii.

In all these cases the paralysis, I repeat, occurs suddenly. But there are instances in which it comes on slowly and by degrees—namely, when it is the consequence of a lesion which affects the facial nerve secondarily, as when some organic alteration in its neighbourhood after a time compresses the nerve in some part of its course, or alters its structure.

You know the course and distribution of the seventh nerve. You know how, emerging from the lateral column of the cord just as this column passes under the pons, it enters the internal auditory meatus, goes through the flexuous canal of the aqueductus fallopii, and comes out of the skull through the stylo-mastoid foramen; and how it then gives off several small branches—the posterior auricular, stylo-hyoidean, and infra-mastoidean—and then divides into two branches, the cervico-facial and temporo-facial. Now, before it enters the temporal bone, and after it has issued from it, this nerve is sometimes involved in tumours, which, whether they be developed inside the cranial cavity or in the region of the parotid, may compress or disorganize it. It is far from being safe from all accident while it traverses the temporal bone; necrosis or caries, and suppuration of that portion of the temporal bone, may bring on destruction of the nerve and, as a consequence, paralysis of the parts which it supplies. Several instances of this have come under my notice—one, among others, in a boy seventeen months old, who died in one of my wards at the Necker Hospital, and whose case was published in the "Bulletin général de Thérapeutique" for January 1847.

From what I have just told you, gentlemen, you may foresee that your prognosis should not be favourable in every case of Bell's paralysis. I will add that, in some very rare instances, this affection is due to a cerebral lesion. Graves states that he has twice seen paralysis *exclusively limited to the face* in small cerebral hæmorrhages; and my colleague in the hospitals, Dr. Duplay, has recorded several cases of the same kind in a very remarkable memoir. Graves makes the remark that paralysis which is thus localized is not very extraordinary, since cerebral hæmorrhage pretty frequently manifests itself only by paralysing the tongue or one arm. I have very frequently met with individuals in whom there had evidently been a very limited extravasation of blood, and whose features were considerably distorted, although they did not complain of weakness in the limbs of the same side. It must be added, however, that when such patients are examined with great care, when they are asked to get up and walk, there may be perceived a certain hesitation

in the movements of their leg of which they are not conscious; and if their strength be tested by means of Burq's dynamometer, it is found that the pressure made on the instrument by the hand, on the same side as the paralysis of the face, is evidently less than that made by the other hand. I am therefore very much disposed to believe that the illustrious Dublin physician has not had recourse to the various tests which I have just mentioned in the case of the two individuals whose history he relates very concisely. As to the instances recorded by Dr. Duplay, they have not convinced me; and it seems to me that in the first two cases, which he gives as typical ones, there had been at separate periods Bell's paralysis and cerebral hæmorrhage, diseases which by no means exclude one another.

But does it never happen that a cerebral lesion produces facial paralysis presenting the characters of Bell's paralysis? There are cases of the kind, as, for instance, in lesions of the pons varolii, as M. Vulpian's experiments have conclusively shown. He found that a very slight wound of the fourth ventricle produced paralysis of the face having all the characters of Bell's paralysis, even those indicated by M. Duchenne (de Boulogne)—namely, the absence of all electric excitability of the muscles supplied by the seventh pair. It is conceivable, therefore, that if a small hæmorrhage occurred in a very limited spot of the pons, it could give rise to the symptoms of Bell's paralysis exclusively. But such cases are so very rare that, in the course of a very long practice, I have not yet met with a single instance of the kind. On the contrary, it pretty frequently happens, as I was telling you just now, that in cerebral hæmorrhage without lesion of the pons varolii, there is predominating paralysis of the muscles of the face, simulating Bell's paralysis. Let us try then and distinguish them.

Now, there is a capital point of distinction of which I have already told you, and on which I cannot insist too much—namely, paralysis of the orbicularis palpebrarum. However complete hemiplegia of cerebral origin may be, I have never seen *complete paralysis of the orbicularis palpebrarum; the eye can always be closed;* whilst in Bell's palsy, paralysis of the orbicularis palpebrarum is never absent, and the eye cannot be completely closed. Dr. Cazalis, physician to the Salpétrière, has like me paid attention to this point of symptomatology, and he declares that he has never seen a single case of cerebral hæmorrhage or softening, in which the patient was unable to close his eye on the affected side, however grave the paralysis might be.[1] Yet, in some exceptional cases, all the branches of

[1 Whenever in hemiplegia of cerebral origin the face is affected, I have found that although the patient can close both eyes simultaneously, he cannot

the facial nerve are not affected (those, for example, which supply the muscles of the eyelids may escape), so that the symptom which I just now pointed out to you is sometimes absent. In such cases one should have recourse to the sign mentioned by Duchenne, to which I alluded just now, and which has been confirmed by Vulpian's experiments. In facial paralysis of cerebral origin, the muscles respond normally to electric irritation, whilst their contractility is not at all or scarcely at all roused by an electric current, if the paralysis be owing to an injury to the seventh pair.

In cases of severe chronic otitis, with destruction of the tympanum and ossicula, it is not uncommon to find the petrous portion of the temporal bone in a great part carious, and to see facial paralysis come on. Whilst I was physician to the scrofula wards in the Hospital for Sick Children, I have often pointed out to the pupils who went round with me the relations between chronic diseases of the internal ear and Bell's paralysis. But the disease does not, unfortunately, confine itself to destroying the facial nerve in its passage through the aqueductus fallopii; it attacks the cranial surface of the petrous portion, and on the pus raising and then perforating the dura mater, abscesses are formed in the base of the skull, and purulent infiltrations of the arachnoid result, to which Abercrombie and Hamilton were the first to call the attention of practitioners. Those are terrible accidents, which perhaps never spare life, as you saw last year so sad an instance in the patient at No. 30 in St. Bernard Ward.

In some cases the pus makes its way even into the spinal cavity; and I cannot help quoting to you, in reference to this point, the history of a boy, ten years old, who was attended by Dr. Graves (of Dublin):

"A boy, about ten years old, was admitted into the Meath Hospital, labouring under general dropsy. He appeared of a scrofulous habit, and was much worn down by long-continued diarrhœa. Under appropriate treatement his symptoms gradually but slowly disappeared, and he was restored to comparative health. We now observed that the right side of the face was affected with paralysis, and on examination found that he had been subject to a discharge from the right ear for seven years previously. The paralysed cheek presented the phenomena usually observed in 'Bell's paralysis.' He was attacked soon

close the eye on the same side as the paralysis while the other remains open. This shows that the orbicularis palpebrarum is to some extent paralysed, because it can only act in combination and simultaneously with the muscle on the opposite side. I am not aware that this fact has been noticed before, but its constant presence in cases of hemiplegia in which the face is involved has struck me forcibly.—Ed.]

after with acute pain in the ear, and in the left side of the head; a fortnight after, convulsions set in; the pain moved from the side to the back of the head, then to the back of the neck, and ultimately extended the whole way down the spine, and about this period the otorrhœa diminished. A few days before death *he was attacked with spasms resembling those of tetanus, and the surface of the body became exquisitely tender to the touch.* He never had any loss of motion, and to the last his intellect was perfect.

"From the period when the pain set in to that of his death, the convulsions returned about six times.

"*Post-mortem.*—The portio dura was dissected on the face, and found healthy; the nerve was also healthy, from its origin at the base of the brain to its entrance at the meatus auditorius; immediately above this opening the dura mater was of a greenish color, detached from the bone, as if by fluid, and perforated by a round hole, large enough to admit a small crow-quill. On dividing this part of the membrane, the space between it and the bone was occupied by a thick, greenish, and offensive pus, and the opening in the dura mater was observed to lie exactly opposite the foramen in the petrous portion of the temporal bone, called the *aqueductus vestibuli;* this opening was much enlarged, and the bone around it was in a carious condition. The nerves at the base of the brain were bathed in this thick green pus, but the organ itself was everywhere healthy, and free from any excess of vascularity. The spinal arachnoid was also filled with the same kind of matter, but the spinal cord itself presented no trace of disease."[1]

The *symptoms* of facial paralysis vary according as the lesion to which it is due is seated at a more or less distant point from the origin of the seventh pair. But, whatever be the seat of the lesion, the patient's physiognomy wears a strange aspect, which is perfectly characteristic.

Even in the state of repose, there is a striking want of symmetry between the two halves of the face, owing to the absence of antagonism in the muscles, which give regularity to the features through their co-ordinate contraction. The sound cheek looks wrinkled and shortened; the labial commissure on that side is drawn outwards and upwards, and is on a higher level than the opposite one. When the paralysis is very marked, the commissure on the affected side remains half-open, and the saliva escapes constantly through it.

Moreover, the cheek is flaccid from paralysis of the buccinator muscle, and yielding in forced expiration to the pressure of the air exerted from within outwards, it swells out and then falls down flapping, like a curtain as it were, in front of the rows of teeth and of the interval between them. Breathing is badly

[1] Graves's "Clinical Lectures," vol. i. p. 569.

performed through the nostril on the affected side, from its no longer opening as it does normally, and remaining more closed than it should be and than it indeed is on the sound side, towards which the tip of the nose is slightly drawn. The eye is, on the contrary, more widely open, although the eyebrow is lowered, from the corrugator supercilii being paralysed and unable to keep it up; the eye looks also larger and more prominent than its fellow. The lower lid is everted and depressed, whilst the upper lid, being now under the influence of its elevator muscle alone, is drawn up and maintained in that position. In a pretty good number of cases, there is a constant flow of tears, and the *epiphora*—which is all the more copious that the irritation of the conjunctiva causes the lachrymal gland to secrete more abundantly—is due partly to the fact that the lower lid no longer forms a canal for the tears, and partly (but chiefly) to paralysis of that portion of the orbicularis which forms Horner's muscle. The lachrymal puncta, which it is the office of this muscle to pull inwards and to make prominent, can no longer assume that position, and therefore no longer receive the tears which do not find their way into their normal channels.

Dangerous consequences to the organ of sight may result, in facial paralysis, from the absence of winking, as sometimes happens in grave fevers from the same cause. For you are aware that three sets of nerves participate in the act of winking:—(*a*) branches of the fifth pair, which are sensory, and give rise to the sensation of the want of winking; (*b*) branches of the seventh which are motor and preside over the contraction of the orbicularis muscle, and consequently over the closure of the eyelids; and (*c*) lastly, branches of the third pair, the motor oculi communis, which presides over the contraction of the levator palpebræ superioris, the muscle which opens the eyes. The use of winking is to protect the globe of the eye against any external injury, and especially to spread over its surface the tears which lubricate the membranes that enter into its composition and preserve their limpidity. Now, as soon as a lesion of the facial nerve brings on paralysis of the orbicularis, the patient can no longer wink, and hence his tears are no longer spread over the surface of the globe of the eye, or are only imperfectly spread: besides, from its remaining constantly open, the eye is exposed to the irritating action of the air, and becomes the seat of inflammation, which increases more or less rapidly. The conjunctiva is red and injected, the cornea becomes dry and opaque, ulcerates, and is then perforated, and the eye is lost, in the same manner as in grave continued fevers. These dreadful consequences are not common, however, in cases of facial paralysis, because, on the one hand, winking is in part supplied by the movements of the eye, which are performed by

its intrinsic muscles; and, on the other hand, the patients instinctively remedy the absence of winking by lowering from time to time with their fingers the palsied lid, so as to rub with it the surface of the eye.[1]

When the patient's face gets animated—when he speaks, laughs, or tries to contract the muscles of his face, the deformity characteristic of facial paralysis becomes considerably more apparent, from the immobility of the palsied side contrasting singularly with the exaggerated mobility of the sound side. The labial commissure on the sound side is pulled upwards and outwards; the nostril rises and opens, the eye can be closed at will, and the forehead thrown into wrinkles; whilst on the diseased side, the labial commissure remains lowered, the nostril closed, the forehead smooth.

When the patient speaks, he has some difficulty in pronouncing labial consonants and vowels. The tongue, however, remains in general free, and is protruded in its normal direction, although it apparently deviates, owing to the normal relations between the two labial commissures and the median line being lost, in consequence of which the apex of the organ seems to point away from the median line on the side corresponding to the paralysis. There are yet cases in which the tongue is paralysed, and really deviates—namely, when the branches which the facial nerve sends to the styloglossus and genioglossus muscles are involved. In such cases there exists, also, a peculiarity which several observers have pointed out, and which I noticed myself in the case of the young man in St. Louis Ward: I mean paralysis of a portion of the velum peleti and uvula, and deviation of the latter. You could see, when you looked into the throat of this patient, that the uvula inclined to the left side (the facial paralysis was on the right side), so that the semi-arch comprised between it and the pillars of the soft palate was markedly much narrower than the semi-arch on the right side.

Paralysis of the tongue and soft palate is an uncommon complication of facial paralysis after all, and can only be explained by admitting that the lesion of the seventh nerve is seated near the origin of the nerve, or at the very least before it bends at the genu, in the aqueductus fallopii; for it is at that part that the nerve gives off the branches which go to the spheno-palatine ganglion, from which proceed the branches destined to the muscles of the soft palate, to the styloglossus, and the genioglossus.[2]

[1 Valleix mentions a case of facial palsy of twenty years' standing, in which there was no alteration of the eye. ("Guide du Médecin Praticien," vol. i. p. 746).—Ed.]

[2 Great diversities of opinion exist as to the position of the uvula and soft

In consequence of the paralysis of the orbicularis oris, the patient cannot perform certain actions, such as that of spitting, or at least he has great difficulty in spitting to a certain distance; and you may remember that this was the first symptom which made the patient in St. Agnes Ward notice the accident which had happened to him. He could no longer whistle, and

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palate in facial paralysis. There can be no doubt that these parts are rarely involved—so much so, indeed, that an eminent authority, Dr. Todd, was disposed to regard any changes in the normal position of the soft palate and uvula in facial palsy as a mere coincidence probably; while in France Landouzy, who has paid special attention to the subject, denies entirely that any change ever takes place. In a good many cases of unilateral facial paralysis which have come under my notice, I have looked in vain for deviation of the uvula and lowering of the soft palate. In one of these the patient, a girl of 18, was affected with well-marked paralysis of the left side of the face, neuralgia of the fifth on the same side, and complete deafness of the left ear—all resulting from repeated attacks of otitis, with chronic purulent discharge from the left ear. Although the lesion of the facial was clearly seated in that portion of its trunk which lies within the petrous portion of the temporal bone, there was not the slightest deviation of the uvula to either side of the median line, and the arches of the soft palate were on both sides of equal height and similar shape. When deviation of the uvula exists, the generally accepted opinion is that the organ is inclined *towards* the sound side, as in the above case of Prof. Trousseau. Thus Dr. Todd says, when speaking of facial palsy: "In some instances the velum of the palate participates in the paralysis, and when you look into the patient's throat, you will find the uvula inclining *away from* the paralysed side, and the velum drawn to the sound side." Hasse, Montault, Debrou, and Longet are of the same opinion. Romberg, however ("Diseases of the Nervous System," Syd. Soc. vol. ii. p. 275), makes a perfectly opposite statement. "Bidder," he says, "was the first to offer a physiological explanation of the functions of the nervus petrosus superficialis major; he considers it as a motor nerve, passing from the facial to the spheno-palatine ganglion, destined to move the velum palati. The unilateral paralysis of the velum palati appears to depend upon the loss of conducting power in those nerve-fibres which give the uvula a slanting direction, causing its point to be directed to the paralysed side: it resumes its normal position after a cure has been effected. In four patients affected with palsy of the facial, I have noticed the hemiplegic condition of the velum palati."

In a very able paper, published in the "Glasgow Journal of Medicine" for August 1865, Dr. Sanders (of Edinburgh) has, however, shown that "the position of the uvula varies frequently, both in the natural and the hemiplegic palate, being twisted sometimes to the right, sometimes to the left—the point directed sometimes to the paralysed, and sometimes to the sound side." He is therefore of opinion that curvature of the uvula, taken by itself, is an uncertain sign, and does not possess the diagnostic importance which has been ascribed to it.

The only reliable sign, according to him, that the palate is involved in lesion of the portio dura, is the existence of a "vertical relaxation or lowering of the corresponding half of the velum palati, with diminished height and curvature of the posterior palatine arch on the paralysed side. This condition is due to paralysis of the levator palati, and probably" (he adds) "of the intrinsic fibres of the velum only—*i.e.*, of the fibres which spread out in a vertical direction within the velum itself; for the paralysis is incomplete, as the palate can be raised *en masse*." Davaine is the only other author who has called attention to this deformity of the soft palate, and he also ascribes it to paralysis of the levator palati. (See his *Mémoire sur la Paralysie générale ou partielle des deux nerfs de la 7e paire*, in *Mémoires de la Société de Biologie* for 1852, p. 164.)—Ed.]

when he attempted to inflate his cheeks, by blowing while his mouth was shut, he could not keep the air in, and it escaped through his half-opened lips.

Mastication is itself impeded. The paralysed buccinator being unable to push back the food into the cavity of the mouth, as it does normally, the food accumulates outside the row of teeth, in the sort of pouch formed by the distended cheek, and the tongue must constantly go there after it. It frequently happens that the patient is even obliged to use his fingers in order to get the food back between his teeth, or he supports with his hand his paralysed cheek while eating, in order to prevent it from getting distended—thus instinctively supplying the place of the muscle which no longer acts.

While motility is thus impaired, tactile sensibility is perfect in the paralysed parts, although it occasionally happens that the sense of taste is perverted on the side of the tongue which corresponds to the motor paralysis. On October 8, 1863, I was consulted by a patient attended by M. Pératé. Two months previously he had got wet through, while riding outside an omnibus. A few days afterwards he went on a railway journey, and the window being down, the left side of his face was exposed to the wind. On the following day it seemed to him, when he ate, that his food tasted (to use his own words) like "salt plaster." After another day, the left side of his face was completely paralysed. The alteration of the sense of taste was still present when I saw the patient, although to a less degree.

Is this perversion of taste a proof that the chorda tympani is a sensory nerve? or is taste modified only because this nerve influences the secretion of saliva, as it has been shown to do by Claude Bernard, and any injury to it causes modifications of that secretion, the utility of which is indispensable for the regular action of the sense of taste?[1]

[1 The experiments of Claude Bernard have clearly shown that the chorda tympani is a *motor*, not a *sensory* nerve; for if it be divided before it joins the lingual branch of the fifth, galvanic excitation of its peripheral end will cause a flow of saliva from the excretory duct of the submaxillary gland, while no such result follows galvanization of the central end of the nerve. Although division of the chorda tympani arrests the secretion of the submaxillary gland, the parotid still continues to secrete; but destruction of the facial nerve at its origin, or at its entrance into the internal auditory meatus, is followed by an arrest of the secretion of both salivary glands. This experiment of the celebrated physiologist accounts, therefore, for the dryness of one side of the mouth, noted in some cases of facial paralysis due to a lesion of the nerve within the cranium. Professor Bernard believes that the influence of the facial is conveyed to the parotid through the small petrosal branch, which goes to the otic ganglion, as neither division of the chorda tympani nor excision of the spheno-palatine or Meckel's ganglion influences the secretory action of the parotid.

In a case of double facial paralysis, the result of an external injury to the head (compression between two beams), which is related by Romberg

Tactile sensibility is not only preserved, but there is also, in some cases, a sensation of pain in the affected parts, due to the rheumatic agency under the influence of which the paralysis set in.

("Diseases of the Nervous System," Syd. Soc. vol. ii. p. 274), there was an unpleasant sense of dryness in the cavity of the mouth, which the patient sought to remove by frequent rinsing and gargling. The lower surface of the tongue and the cavity of the lower jaw were drier than usual. The parotid still continued to secrete, however, and the patient did not complain of dryness in the part of the oral cavity corresponding to the opening of the ductus stenonianus. In this case, the arrest of the submaxillary secretion, with persistence of that of the parotid, may be explained by the injury to the facial nerve being situated below the origin of the small petrosal, but above that of the chorda tympani.

As to the diminution or perversion of taste after division of the chorda tympani, Prof. Claude Bernard suggests (but with hesitation) that it may be brought about in two ways: either through a modification of the circulation of the part, or through deficient erection of the papillæ of the tongue, preventing proper contact between them and the sapid substances. (Claude Bernard, *Leçons sur le système nerveux*, vol. ii. p. 174.)

See also a most able memoir by this learned physiologist: *Sur la couleur du sang veineux des glandes*, in Brown-Séquard's *Journal de la Physiologie de l'homme*, etc. (vol. i. p. 233), in which he discusses how and by what process division of the chorda tympani influences the secretion of the submaxillary gland. It is not, as Arnold and Romberg seem to believe, by paralysing the excretory duct of the gland, and thus preventing the evacuation of the secretion, but by interfering with the actual secretion itself. Thus Bernard found that the blood circulating in the veins of the gland is of a black colour, while the secretion is extremely diminished in quantity, and is thick and viscid, when the chorda tympani has been divided; but that on galvanizing the peripheral end of the nerve, the venous blood becomes of a bright-red colour, and saliva is abundantly secreted.

Another sense besides that of taste is sometimes affected in cases of facial paralysis, namely the sense of hearing, which becomes more acute. Dr. Landouzy (of Rheims) was the first to draw attention to this symptom. (*De l'exaltation de l'ouïe dans la paralysie du nerf facial*, in *Union Médicale* 21 *décembre* 1851.) The explanation which he suggests is that there is increased vibration of the tympanum, which is relaxed in consequence of the paralysis of its tensor muscle. In a recent case of facial palsy, he found that the patient complained of pain in the ear when a pistol was fired by his side, while he had no such sensations when his face was galvanized, so as to make the muscles contract, and the tensor tympani with them. Dr. Brown-Séquard suggests, however, a different explanation. Division of the sympathetic in the neck, he says, and avulsion of the facial nerve, seem to be both followed by abnormal acuteness of hearing. But in the case of the sympathetic, the exaggerated sensitiveness of the organ of hearing is not owing to paralysis of the tensor tympani muscle, but to hyperæsthesia of the auditory nerve consequent on paralysis of its bloodvessels; and, according to this great physiologist, the hyperacuteness of hearing in facial paralysis is probably dependent on the same cause. (*Journal de la Physiologie de l'homme et des animaux*, vol. i. p. 429.)

In a case of facial palsy which came under my care within a fortnight of the setting-in of the complaint, hearing was much more acute on the paralysed side, and the patient could hear the ticking of a watch at a much greater distance from that side than from the other. But this symptom is not constant, for it was absent in another recent case of facial palsy which I saw some time ago; while for the first week after the paralysis set in, the patient spontaneously complained of not being able to taste on the same side as the paralysis, and of that side of her tongue feeling "as if it had been scalded."—Ed.]

It seems, gentlemen, that there can be no possibility of error in a case of facial paralysis, or that the whole question of *diagnosis* consists merely in investigating the causes which brought on the complaint. Yet the case of a young woman, lying in bed No. 7, St. Bernard Ward, has shown you that this diagnosis was not always so simple as one would imagine. You remember how the patient to whom I allude was admitted into the Hôtel-Dieu for a puerperal affection, into the history of which I need not go here, and which was besides of no gravity. From the first day I saw her, however, I was struck with the deformity of her face, which, at first sight, suggested the idea of paralysis of the *left* side of the face; for her face was distorted, and deviated notably to the *right*. The upper lip and the ala nasi of that side were drawn upwards; the labial commissure was pulled upwards and outwards; the naso-labial sulcus, which was also pulled upwards, was deeper than normal, whilst the corresponding nostril was less open than the other. Yet the eye on that side looked larger than the left eye; the under-lid was depressed, and slightly everted; and the tears, which were abundantly secreted (especially when the patient had looked at some object), flowed over the cheek instead of through their normal channel: at such times, also, the sight was somewhat obscured.

On carefully examining the patient's face, one was not long before noticing that there occurred, on the right side, slight convulsive movements, analogous to those which characterize spasmodic tic. Those movements were spontaneous, but could also be induced by rubbing the cheek or the upper lip with the tip of the finger or a penholder, or by gently tickling the skin of those parts.

If left facial paralysis was thought of at first sight, the depression of the lower lid, and the less marked expansion of the nostril on the right side, were already sufficient to cause a modification of the diagnosis. But when the patient attempted to move that side of her face, there could no longer be any hesitation, and it became manifest that it was the right side which was affected. When she spoke, and still more when she laughed, her face was pulled with force to the *left*, the upper lip and the ala nasi on that side going obliquely upwards, and the labial commissure being drawn with considerable energy upwards and outwards. When she attempted to blow, her left cheek swelled out, and her mouth remained closed on that side; whilst her right cheek was flaccid, and her mouth opened out a little on that side. Besides, she could not shut her right eye, however much she tried.

She gave us the following account of her case:—She had had complete paralysis of the *right side of the face* eight years previously. It had set in suddenly, subsequently to a *cold* caught

during a walk by the seaside, after she had had a tooth extracted. For eight months the application of leeches and other therapeutic measures were vainly tried against this affection, which was accompanied by violent pain in the head, and which yielded at last under the influence of a treatment by localized electricity carried on for four months. She seemed to be radically cured. Her features had completely recovered their regularity, when the new change, which attracted my attention, occurred, and which the patient stated she had perceived of late only.

Several medical men, whom she had consulted since then, mistook, not the nature of the disease, but the seat of the paralysis, placing it on the left, whilst it was undoubtedly on the right; for no one among you can doubt that we have, in this case, to deal with *convulsion* and *contraction of the muscles of the face*, consecutive to paralysis.

We could here suppose, gentlemen, but could not affirm, the existence of a relation between the facial paralysis and the convulsion of the muscles supplied by the seventh pair, although this convulsion might well be a coincidence only. Indeed, what Graves has called *spasm of the portio dura of Bell*, or, in other words, spasm of the facial muscles, occurring independently of all painful affection and of all paralysis, is pretty common; and Graves relates a very curious instance of it in his thirty-eighth lecture. I have often seen it myself—generally, it is true, in connection with neuralgia of the fifth pair, with that variety of neuralgia which I have termed *epileptiform*, and of which I have spoken to you at length.

Simple contraction of the muscles of the face is very common after Bell's paralysis. In the case of the young woman to whom I alluded just now, it was partial, as it most commonly is; the raised upper lip and ala nasi, and the deviation of the corresponding labial commissure, indicated that the contraction only involved the orbicularis oris, the zygomatici, the buccinator, and the levator alæ nasi et labii superioris; whilst the depressed lower lid, the less expanded nostril on the right side, showed also that the orbicularis palpebrarum and the dilator muscle of the nostril (transversus pinnæ) were still paralysed. The contraction was, besides, mixed up with some degree of paralysis, as was shown by the want of power of contracting at will the affected muscles.

I have often already, and for a long time past, called your attention to the contraction of the facial muscles which follows Bell's paralysis. There then occurs a process analogous to what we observe in other muscles in cases of hemiplegia due to cerebal hæmorrhage or softening. As I have had occasion to tell you, when the hemiplegia has been such as to abolish all movement for several weeks, it rarely happens that the muscles

of the arm and forearm do not become contracted irremediably. If you visit hospitals for the aged, you will be struck with the extreme frequency of this affection. The forearm, in such cases, is half-flexed on the arm, the hand on the forearm, and the fingers (more particularly the two last phalanges, and the ungual phalanx of the thumb) are forcibly bent into the palm of the hand. The contraction is sometimes a little painful, and attempts can never be made to overcome it without causing acute pain, and the same result follows when the muscular masses suffering from this spasm are firmly compressed. Contraction, following paralysis, is therefore of extremely common occurrence, and it is perfectly natural that it should come on after Bell's paralysis, when this affection has been carried to an extreme degree, and has lasted a long time.

There was last year in my wards, if you recollect, another very striking instance of this. Of course all the cases are not exactly alike—that is to say, one muscle will be at one time contracted, and another muscle at another time. In one patient, the orbicularis palpebrarum will be affected, and the consequence will be that the eye, instead of being more open than the other, will close and look smaller; in another, as in our young woman, the buccinator and the zygomatic will be contracted. It may also happen that the muscles become shorter in course of time, in which case there will not only be a simple deformity of the face, but also a considerable impediment in its mobility. This contraction of the muscles of the face is, I repeat, a frequent termination of the so-called rheumatic paralysis of the seventh pair. Dr. Duchenne, in his treatise on *Localized Electrisation*, has devoted an interesting chapter to this subject.[1] I am the more surprised at our classical works making so little mention of it, that muscular contraction, sequential to paralysis of the limbs or trunk, is a symptom which has been universally indicated.[2]

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[1] In several cases of contraction of the facial muscles subsequent to paralysis which have come under my observation, I have been enabled to verify the accuracy of Dr. Duchenne's statement, that the state of contraction is preceded by spasm of the paralysed muscle under the influence of artificial excitation. Another sign of threatening contraction, given by this acute observer, is a rapid return (in less than a fortnight) of tonicity in a paralysed muscle, the electro-muscular contractility of which had been completely abolished. (*De l'électrisation localisée*, 2e éd. pp. 677, 678.)—Ed.]

[2] The following case is a good illustration of the occasional difficulty which is experienced in determining which is the side of the face that is paralysed: —A tall healthy-looking woman, M. O., an ironer, aged 64, came under my care in October 1864, at the National Hospital for the Paralysed and Epileptic. The two sides of her face were not symmetrical, the right looking smaller than the left. On the former side (the right) the forehead was marked by persistent horizontal as well as vertical wrinkles; the orbicularis palpebrarum kept acting spasmodically, causing rapid involuntary closure of the eye; the tip of the nose was drawn to the right side; the sulcus between the ala nasi and the

It was necessary to fill up this omission in the history of facial paralysis, in order to put you on your guard against possible errors of diagnosis. A little care will be sufficient to make you avoid them. As to the differential diagnosis of the various kinds of paralysis from one another, it should be based on a knowledge of the circumstances in which the complaint set in, of the course of its development, and the concomitant phenomena.

In one of our previous conferences, I have dwelt long enough on the differential characters of Bell's paralysis, and of facial paralysis symptomatic of a central affection such as hæmorrhage, and I need not return to the subject now. There are embarrassing cases, however—namely, when the facial paralysis is due to a tumour of the brain, developed either in the meninges, or in the substance of the organ itself, or in the petrous portion of the temporal bone, in the neighbourhood of the spot where the seventh nerve enters the aqueductus fallopii. The cause of the paralysis, especially when it sets in suddenly, may be mistaken, and it may be thought of a rheumatic nature. Such cases are, fortunately, very rare; and other phenomena enable one besides to make, before long, a correct diagnosis.

Idiopathic facial paralysis generally gets well, and all the more

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angle of the mouth was considerably deepened; the angle of the mouth was pulled upwards; the under-lip was slightly everted, and the chin considerably wrinkled. The sensibility was normal, and no pain was complained of on this side.

On the *left* side, on the contrary, the forehead was smooth, and the patient could not frown; the eye could be imperfectly closed, the lower eyelid was everted, and there was some epiphora in consequence; the eye looked larger than on the other side. When asked to inflate her cheeks, the right one scarcely bulged while the left swelled out feebly, and then flapped down as the air escaped out of the left corner of the mouth. The muscles of the left angle of the mouth and the left half of the orbicularis oris could not be contracted at will, and the patient could not articulate the labial consonants, such as *b* and *p*. Whenever she drank, the liquid ran out of that side of the mouth; and when she ate her food, she had to put her hand to that side, in order, she said, to keep the food in. She complained of soreness of the left cheek and inside the left eye, and of dryness of the cavity of the mouth. She could taste equally well on both sides of the tongue; her uvula was slightly inclined to the left side, and there was depression of the left half of the velum palati. The sense of hearing was more acute on the *left* than on the *right* side, for the patient could hear the ticking of a watch at a farther distance from the left than from the right ear.

The left side of the face had been affected for the last fortnight only, while the patient had been worried for six years with the spasms on the right side.

From this history, and from the actual condition of the parts, there could be no doubt that the poor woman (who, from the nature of her occupation, was exposed to draughts of cold air—whenever a door, for instance, was opened leading into the hot close room in which she worked as an ironer) had been seized with facial paralysis, on two separate occasions—on the right side six years previously, and on the left a fortnight before she came under my observation. The persistent contraction of some of the muscles of the right side of the face, and the spasmodic action of others, had supervened on paralysis.—Ed.]

rapidly that it set in suddenly, and the patient is young. There is an important point which you must know, however—namely, that under certain circumstances the complaint stubbornly resists all treatment, although nothing in the phenomena which characterize it gives you a clue to this; whilst in other cases, presenting identical symptoms, the disease yields with the most marvellous facility. Dr. Duchenne (de Boulogne) has shown that localized electrisation affords us a means of distinguishing such cases, abolition of the electric contractility of the palsied muscles being regarded by him as a certain sign of the incurability of the disease.

Now, gentlemen, a few words as to treatment. Above all, do not forget that facial paralysis is sometimes such a transitory complaint that it gets well in 24, 15, and even 12 hours, before medicine has had time to interfere. Such cases are exceptional, however. Antiphlogistics, leeches, and cupping in front of the ear, and on a level with the mastoid process, are indicated when the presence of pain, and a certain amount of swelling of the region about the parotid, seems to point to an irritation of those parts.

When the disease is of a less acute character, remedies which stimulate the skin should be had recourse to, and, of these, blisters rank first. If they fail, more energetic measures are called for, such as transcurrent cauterization, cauteries, and moxas.

I have obtained pretty good results from the use of preparations of strychnine or of veratria, by the endermic method. I have the raw surface of a blister dressed with from 2 to 10 milligrammes ($\frac{1}{25}$th to $\frac{1}{5}$th of a grain) of sulphate of strychnine or of veratria, which are always mixed with five or six times their weight of powdered sugar. I have also seen some good done by the application, on the region of the parotid, of compresses steeped in tincture of nux-vomica. Lastly, acupuncture, electropuncture, or electrization simply, have been found useful; but it should be remembered that faradization should be used according to certain rules, well laid down by Dr. Duchenne.[1]

[1 Duchenne (*De l'Électrisation Localisée*, p. 687) recommends that each facial muscle should be separately galvanized, instead of passing the electric current through the facial nerve. The reason which he gives, in addition to the fact which he has proved that paralysed muscles are more powerfully influenced in their nutrition when directly galvanized than when a current is transmitted through their nerve, is that the muscles regain voluntary contractility at different periods, some before others: hence the necessity of galvanizing most those which have recovered their contractility least. He lays great stress on the importance of using an apparatus capable of producing a current with very rapid intermissions. When the muscles have begun to *contract*, however, he recommends that the intermissions should be *few*, and the *sittings* short and far

It is not necessary for me to add, that all I have said on treatment refers merely to the so-called rheumatic paralysis, for it is self-evident that the paralysis caused by an accidental division of the nerve,[1] or its destruction in diseases of the petrous portion of the temporal bone, is quite beyond the resources of art.

As yet, I have only spoken of facial hemiplegia; but before concluding, I will say a few words about *double facial paralysis*, an affection which is not even mentioned in the treatises on medicine and surgery which are in your hands. Dr. Davaine, who has the merit of having brought together, in a long and important memoir, cases of this kind scattered through scientific records,[2] has summed up its characters. They vary according as the paralysis is general or partial, complete or incomplete.

In the general and complete variety (the only one of which I shall speak here, for it is the only one which has been observed in man, partial paralysis of both facial nerves having been met with in the lower animals alone), the features have not lost their regularity, or, more properly speaking, there is no longer that want of symmetry which in the hemiplegic form arises from the absence of antagonism between the muscles of the affected side and those of the other. The motionless face assumes a peculiar aspect, and looks like a lifeless mask on which the impressions of the soul are no longer expressed but by changes of colour. The forehead is smooth, the superciliary region lowered;

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between; otherwise, the contracted state of the muscle will be increased, and rendered probably irremediable.

When the muscles are firmly contracted, he advises that they should be pulled out and stretched mechanically in the direction of their fibres; and to remedy contraction of the buccinator, he recommends to keep a small billiard-ball inside the cheek, which is after a time replaced by a larger one. This treatment should, of course, be continued perseveringly for a long while. In one case in which I tried the pulling and stretching plan, considerable improvement followed. When the deformity is so considerable as to produce a very ugly appearance of the face, or to impede the movements of the jaw in mastication, subcutaneous myotomy might be had recourse to. Romberg states that myotomy has been proposed and even practised by Dieffenbach in cases of facial palsy of old standing, attended with considerable deviation of the face. But the muscles which the German surgeon divided were those on the healthy side—a procedure which seems certainly illogical. When only one or two muscles are contracted, Duchenne suggests a very original plan, which he has, he states, put successfully in practice in one case. It consists in passing an induced current, with very rapid intermissions, through the healthy antagonistic muscle, and thus bringing it into a condition of exaggerated, persistent tonicity.—Ed.]

[1] Romberg mentions, however, the case of a boy, nine years old, who had been attacked by murderers, and had received a penetrating stab with a knife in the vicinity of the stylo-mastoid foramen, and in whom the paralysis of the labial and nasal branches of the facial disappeared after two months.—Ed.]

[2] Mémoire sur la paralysie générale ou partielle des deux nerfs de la septième paire. (Mém. de la Société de Biologie, 1852, et *Gazette Médicale de Paris* 1852 et 1853.)

the eyes are wide open and cannot be closed; the lower lid is half-depressed, and, as in the hemiplegic form, the tears flow constantly over the cheeks, while the half-opened lips allow the saliva to run out of the mouth. The nostrils, already diminished in calibre, fall in still more during inspiration, while in forced expiration the cheeks are puffed out, soon to sink in again like loose sails. The other symptoms which I mentioned to you when speaking of facial hemiplegia—namely, the difficulty of mastication, the inability to spit out, to whistle, or to blow, the difficulty in pronouncing certain consonants and the labial vowels—are much more marked in double facial paralysis; the voice, besides, has a nasal twang, because the soft palate, which is sometimes involved, as we have seen, in facial hemiplegia, is generally much more completely so in the double form of the disease. On looking down into the patient's throat, there is no deviation of the uvula as in hemiplegia, nor diminution in the diameter of one of the arches comprised between the uvula and the corresponding pillars; the two arches are symmetrical. But this complete paralysis of the soft palate causes, in addition to the nasal twang of the voice, difficulty of deglutition, and the return through the nose of the liquids drunk. The difficulty of deglutition is due to other causes also—in part to the paralysis of the posterior belly of the digastric and of the stylohyoid muscles, which are supplied by a branch of the facial nerve, and the latter of which raises the base of the tongue; and in part to the paralysis of the pharynx itself, which is also supplied by branches of the seventh pair: lastly, the tongue can no longer be protruded out of the mouth with ease, nor its tip curved upwards.

You understand, gentlemen, that it is impossible to speak in general terms of the course, the duration, and the termination of double facial paralysis, because these are, in fact, necessarily subordinate to the *causes* which produce it.

These *causes* are sometimes lesions of the nervous centres, such as extravasations, softening, &c., the symptoms of which are limited to the muscles supplied by the nerves of the seventh pair, as in one of the cases recorded by Dr. Davaine, although we find it impossible to explain by the anatomical lesions why the paralysis was so localized.

Sometimes the cause is some affection involving the two facial nerves in their course through the petrous portion of the temporal bone. Thus, Dr. Davaine relates an instance of double facial paralysis resulting from a violent concussion, which had fractured both temporal bones at the same time. Now, in such a case, the paralysis is explained by compression or laceration of the nerves. But one may conceive how a morbid influence capable of acting on several organs at the same time,

and especially on the bones, such as scrofula and syphilis, will be more liable than any other to produce a simultaneous lesion of the two temporal bones, and thus to bring on double facial paralysis. The author, whom I mentioned just now, borrows from Sir C. Bell a case observed by Dupuytren—namely, that of a girl, sixteen years of age, who had double facial paralysis, beginning on the left side, and involving the right a week afterwards, and which disappeared under the influence of an anti-syphilitic treatment carried on for eight months. I read, a few days ago, in the "France médicale," a similar case extracted from the "Dublin Quarterly Journal," and published by Dr. O'Connor. The patient had for a long time exhibited symptoms of constitutional syphilis, and was particularly suffering from periostitis of the cranial bones. The facial paralysis in this case also showed itself first on the left and then on the right side. Hearing was not impaired, and there was no disturbance of the intellect, although, judging only from his aspect, the patient looked a perfect idiot. The features were expressionless. The eyes were constantly staring, injected, red, and bathed in tears, which kept dropping on the cheeks. The flaccid and hanging commissures of the lips allowed the saliva to escape, as well as any liquids which the patient attempted to swallow, deglutition being performed with considerable difficulty only. As the lips could no longer be used for articulation, the voice was guttural, and seemed to issue from the bottom of the throat. As the patient's life was not in danger, the expression of his face excited laughter more than a feeling of pity, so that the jokes of his companions made him leave the hospital, and Dr. O'Connor was not able to find out how the disease terminated.

A third class of causes of this double facial paralysis includes those which act on the nerve as it issues from the stylo-mastoid foramen and on its peripheral ramifications—such, for instance, as cold,[1] and compression by the forceps at the time of delivery.

Before leaving this subject I must call your attention to the

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[1 In a recent number of the *Medical Press and Circular* (December 19, 1866), there is an account of a very interesting case of double facial paralysis, of which cold seems to have been the determining cause. The patient, a man of fine vigorous form, aged 28, and previously healthy, lay all night on a damp floor, after a drinking bout, exposed to a severe draught blowing on his face. When he came to himself the next day, he found that the right side of his face was paralysed, and in less than a week subsequently, the left side became affected in the same manner. He now became so seriously alarmed that he requested and obtained admission into the Richmond, Whitworth, and Hardwicke Hospitals (Dublin), when he was placed under Dr. Lyon's care.

In about three weeks, the left side of the face, that which was the last affected, began to improve, and finally got perfectly well; but the right side proved more obstinate and rebellious to treatment, and although it had regained some power at the end of two months, it had not then perfectly recovered.—Ed.]

fact that double facial paralysis has been confounded with another variety of incomplete paralysis of the face, which I have called *glosso-laryngeal paralysis*, and which has been described by Dr. Duchenne (de Boulogne) under the name of *Progressive muscular paralysis of the tongue, soft palate, and lips.*

You no doubt remember how carefully I took notes of the cases of five individuals suffering from this affection, and who remained for several months in St. Bernard and St. Agnes Wards. These patients had paralysis of the soft palate, of the tongue and the lips; the articulation of certain words and of certain letters, the deglutition of saliva and of food, had at first been difficult and then impossible; but the paralysis had never spread to the upper half of the face. The muscles which are concerned in the act of laughing and in the closure of the eyelids had preserved all their contractility; and even a few moments before their death, from asphyxia brought on by the paralysis of their respiratory muscles, or by the arrest of a bolus of food in the last portion of their pharynx, these patients could still show by the expression of their face their gratitude to those who attended them.

In double facial paralysis, on the contrary, the mask is dumb, and hence the persistence of contractility in the upper half of the face, in cases of glosso-laryngeal paralysis, would alone be sufficient to save one from an error in diagnosis. I may add that in Bell's paralysis the tongue is never paralysed to such a degree as to be incapable of being protruded out of the mouth. And if the patients speak with difficulty, it is less their tongue than their lips which fail in the articulation of words.

Dr. Davaine, however (at a time, it is true, when glosso-laryngeal paralysis had not been yet described), mistook that affection for double facial paralysis, and his memoir contains two cases of it (Cases VII. and VIII.). I confess that a mistake may easily be made, because in that singular partial paralysis the orbicularis oris and the tongue can scarcely move; and as in the act of speaking the movements of the mouth are of necessity most frequently repeated, and as most of the facial muscles converge towards the mouth, the patient suffering from this complaint seems to wear a motionless mask, as if he had double facial paralysis. On looking closely at him, however, it is ascertained that the orbicularis palpebrarum and the other muscles of expression have retained all their energy, while this is not the case in Bell's paralysis. Moreover, the muscles invariably retain their electric excitability, whilst in Bell's paralysis this property is abolished or nearly so.

Dr. Davaine gives, after Marshall Hall, a test by which it may be ascertained whether the cause of double facial paralysis is seated in the brain or in the course of the nerves. In the former case the conducting power of the nerve-trunks is retained for

an indefinite period, so that by galvanizing the trunk and the principal branches of the facial nerves, all the muscles supplied by them are thrown into contraction, as if the muscles themselves were being galvanized; whilst, when the paralysing cause is in the course of the nerves, they very easily lose their conducting power. Moreover, if reflex movements be seen in the paralysed muscles, it will be a sure proof that the cause of the paralysis is in the nerve-centres.

When once the seat of the paralysis has been determined, and its cause known or suspected, they will indicate the proper treatment which should be followed, and I need not repeat what I have already told you when speaking of facial hemiplegia.[1]

[1 Some very curious facts have, within the last few years, been made out respecting the effects of an *interrupted galvanic current* (from a continuous current battery) in facial paralysis. Baierlacher, Schulz, and Meyer, have published cases, showing that palsied facial muscles, which do not respond to a powerful induced current, may be made to contract under the influence of an interrupted galvanic current. The contractions occur when the circuit is closed and when it is opened, but more powerfully at the former than at the latter moment. Their observations have been confirmed by Ziemssen (*Die Electricität in der Medicin.* Studien von Dr. Hugo Ziemssen, Berlin, 1864), so far as the phenomenon itself is concerned, but whereas Baierlacher, Schulz, and Meyer speak of the good effects of an interrupted galvanic current, and of its power of restoring in the palsied muscles the property of contracting under the influence of faradization, Ziemssen records a carefully-observed case of facial paralysis, in which no such result was obtained, after the use for eleven weeks of an interrupted galvanic current.

My friend, Mr. J. N. Radcliffe, Medical Superintendent of the National Hospital for the Paralysed and Epileptic (who was the first to draw my attention to this subject), obtained the same negative results as Ziemssen, in a case of paralysis of the portio-dura on the left side, of twenty-nine days' date, occurring in a young woman from exposure to a cold wind. The palsied muscles were submitted to the influence of an interrupted galvanic current twice a week, for the space of six or seven weeks, but without any good result. Nay, more, contraction supervened while this treatment was being carried on.

I have at present (December, 1866) under my care, at the Hospital for the Paralysed and Epileptic, a young unmarried woman, aged 23, who is affected with paralysis of the right side of the face. The complaint set in a month ago, a few hours after she had got wet through on being caught in a shower. The contrast between the effects of faradization and those of an interrupted galvanic current is well exhibited in her case. For whilst a powerful induced current obtained from a Stöhrer's two-celled volta-electric machine can scarcely excite feeble contractions in the muscles about the right angle of the mouth, and none in the other muscles on the right side of the face, an interrupted galvanic current (from a Muirhead's battery of fifteen cells only) makes all the palsied muscles contract energetically at the moment of closing, and less powerfully at the moment of breaking the circuit. It is a very remarkable circumstance that the same current has no effect on the muscles of the sound side.

As yet, the cases in which the interrupted galvanic current has been used are too few in number to admit of positive conclusions being drawn from them. Mr. J. N. Radcliffe, who is at present engaged in studying the effects of this novel application of galvanism, is giving greater extension to the subject than the authors whom I have already named, by trying it in cases of paralysis with

loss of muscular contractility under the influence of faradization, such as lead-palsy, infantile paralysis, and palsy localized in certain muscles from injury to their nerve or nerves. His observation confirms Schulz's assertion, that when an improvement follows the use of the interrupted galvanic current, the number of cells from which the current is obtained must be gradually increased, as the muscles become less susceptible to the influence of the same current. Such, at least, was the fact noted in a case of paralysis of the deltoid, treated in this manner.

When submitted to the influence of an interrupted galvanic current, palsied muscles contract with considerably greater energy when one of the electrodes is gently moved along the surface, in the same manner as when the electric brush is used in faradization, than when contact is made or broken by simply lowering or raising one of the electrodes. In the former case, the current is irregularly interrupted, and its tension varies, according to Ziemssen, while in the latter, the interruptions are regular, and there is no variation of tension. The former constitutes Remak's "labiler ströme" ("labile" current), and is stated by him to differ from the ordinary induced current, in that the latter decreases gradually from a maximum to nil, while the "labile" current never falls down to nil. According to Heiderhain, however, quoted by Ziemssen, this difference would seem to be illusory. Mr. Radcliffe holds that the difference of action resulting from the mode of applying the electrodes, arises from the *duration* of the application, for "with the same force that the 'labile' current produces energetic contraction, a less marked contraction occurs when the circuit is broken by simply lifting one of the electrodes, whereas, when interruption is made instantaneous by an interrupting wheel, no contraction follows, or an exceedingly slight one."

Dr. W. A. Hammond, of New York, has recently published three interesting cases of infantile paralysis, in which the application of an interrupted galvanic current was productive of great benefit. (Half-yearly Abstract, London, January—June, 1866.)—Ed.]

LECTURE X.

CROSS-PARALYSIS, OR ALTERNATE HEMIPLEGIA.

In most cases it is owing to a lesion of the pons varolii, but it is not an absolute sign of such lesion. It should not be confounded with glosso-laryngeal paralysis.

Gentlemen,—When an individual is struck down with hemiplegia, the paralysis affects the limbs and the face on the same side. There are, however, exceptions to this general rule; and for those rare cases in which the face is paralysed on one side, and the limbs on the opposite side, Dr. Gubler has proposed the name of *alternate hemiplegia*.

Very recently, in September 1861, I saw, in consultation with my colleague and friend, Dr. Hillairet, a little girl from Clermont-Ferrand, seven years old, who had met with a severe fall backwards a few months previously, and had knocked the back of her head and the upper part of her neck against a piece of furniture. She complained almost immediately of heaviness of the head and of feeling drowsy, but after a few days she felt well again. Shortly afterwards, however, she complained of a pain both in the occiput and the forehead. Her friends noticed also that she hesitated in her gait, and that she had grown irritable and made grimaces.

Three months after the occurrence of the accident, there was found weakness in the whole left side of the body, as well as a very marked paralysis of the right side of the face. She was then sent to Paris by Dr. Bourgard, and on our seeing her we thought that the pons varolii and the upper part of the medulla oblongata were injured, and we gave an unfavourable prognosis.

We lost sight of the patient, so that we could not verify the accuracy of our diagnosis; but we thought we could make it in this instance, on the ground of its analogy to the cases recorded by Dr. Gubler, showing the relations which generally exist between cross-paralysis and injuries to the pons varolii.

The body of a woman, however, who died at No. 6 in St. Bernard Ward, of some cerebral affection which had produced cross-paralysis, was examined, after death, in your presence. But the results of the autopsy disappointed us, and seemed in contradiction to the law laid down by my learned colleague of the Beaujon Hospital.

The patient was a servant thirty years of age. She was

admitted into the Hôtel-Dieu for a violent pain in the head, which only dated a few days back, and was not localized in any one spot more than another. She was not feverish, her appetite was good, and she complained of nothing else but the headache. Her menses had come on a few hours before admission, and she stated that she was usually liable to this pain in the head at her menstrual periods.

The next day, by the time I went round the wards, nothing fresh had occurred to call for my attention; yet, although the patient gave clear answers to the questions put to her, I noticed that she laboured under a certain amount of hebetude, of mental languor, which could be after all accounted for by the cephalalgia.

The absence of all febrile symptoms, the good condition of her functions in general, did not call for active medical interference; and I had decided on waiting before adopting any treatment, when fresh symptoms manifested themselves during the day, which towards evening alarmed my clinical assistant. The patient had been suddenly paralysed. There was incomplete motor paralysis of the *right* arm and leg, while tactile sensibility was preserved. When the palsied limbs were pinched or merely tickled, the patient drew them away, although less easily and less quickly than she withdrew her left arm and leg under the same circumstances. The head was inclined to the left, and the face turned to the right, from the contraction of the left sterno-cleido-mastoid, whilst the analogous muscle on the right side was relaxed. There was hemiplegia therefore, but the paralysis, while involving the *right* limbs and the *right* half of the trunk, affected the *left* side of the face. The face wore a singular expression, and was dragged to the right, that is, to the same side as the paralysis of the limbs. The mouth was distorted, the labial commissure on that side was higher than the other, whilst the left cheek was more flaccid than the right. Moreover, the patient, who gave distinct answers to questions put to her, stated that she could not see with her *right* eye, whilst on the *left* side her sight was good; both pupils were equally contracted. The paralysis of the left side of the face, which coincided with the diminution of sight on the right side, was evidently less marked than the paralysis of the limbs.

The pain in the head was as violent as ever, and there was no fever. The patient asked for food, although her tongue had a yellowish coating of fur. An emetic was ordered, but on the following morning the symptoms had become more marked. Motion was more impeded and sensation duller than on the preceding day. The paralysis of the face, although less marked than that of the limbs, had increased, although it was not so marked as in cases where it is due to a lesion of the seventh

pair exclusively. The intellect was impaired, and although the patient was awake, and seemed to hear when she was spoken to, she no longer answered. Death took place at 4 o'clock the next morning.

A post-mortem examination was made about thirty hours afterwards. On removing the calvarium, a pretty considerable quantity of black blood escaped from the gorged vessels of the pia mater over the whole surface of the hemispheres. The congestion was most marked at the base of the brain, and there was found in the interpeduncular space a black mass, consisting not only of vessels distended with blood, but of extravasated blood also, which was in part liquid, and in part coagulated, and had made its way into the fissure of Bichat. The nerves of the seventh pair exhibited no alteration at their superficial origin, behind the pons varolii, although the *right* nerve seemed to tear more easily than the other. Independently of the *meningeal hæmorrhage*, the brain was softened in its central parts, on the left especially, where the corpus callosum, the fornix, and the septum lucidum were broken down when a small stream of water was poured upon them. There was no effusion into the interior of the ventricles; and, lastly, the pons varolii presented no lesion, either on its surface or more deeply; on making numerous sections through it, no tumour was found, nor traces of hæmorrhage or of softening.

The results of this examination, then, as I have told you already, are in contradiction to what Dr. Gubler has taught us on the relations between alternate hemiplegia and lesions of the pons. For in this case—which was, it is true, anomalous (since the phenomena observed during life did not correspond regularly with the organic alterations found after death)—the pons presented no appreciable sign of disease, however carefully we examined it. As no such lesions were found, it has been doubted by some that there had been cross-paralysis; and it has been asked whether I had not made a mistake as to the side of the face which was paralysed, and whether I had not mistaken contraction of the muscles of the right side of the face for paralysis of the left side. The objection, I admit, was all the more founded that the softening of the right facial nerve did not harmonise with the retention of motor power on the corresponding side of the face. But my answer is, that however obscure and inexplicable the facts may be, I have no doubt in my own mind that the case was not one of contraction of the right side, but of left facial paralysis (the left cheek being more flaccid than the right), and that this coincided with paralysis of the right limbs; lastly, that whatever the other lesions of the brain might be, the pons varolii showed no trace of disease.

Although I admit that a rigorous conclusion cannot be drawn from an exceptional case, full of anomalies and of obscurity, it would yet seem that the law laid down by Dr. Gubler is not so absolute as he has asserted. Those among you who have read the two interesting memoirs which he has published on this subject,[1] know that he regards cross-paralysis as a sign of disease of the pons; and, localizing still more specially the seat of the anatomical change, he places it in the bulbous portion of the pons. Hence, this particular form of hemiplegia is, according to him, explained in the following manner: As the lesion involves the facial nerve after its decussation, the face is paralysed directly, while the parts that are supplied by nerves from the spinal cord are paralysed in a crucial manner, the decussation of the anterior pyramids taking place below the pons only.

I am far from denying the value of the reasons urged by Dr. Gubler in support of his position. The cases which he brings forward, and discusses with great talent, are sufficiently imposing in number and of undoubted value; but yet I cannot refrain from thinking that the law which he has laid down is too absolute. Setting aside the case of the woman in St. Bernard Ward, I shall find in Dr. Gubler's memoirs themselves arguments in favour of my opinion; for when he comes to the differential diagnosis of cross-paralysis, and what he terms false cross-paralysis (namely, cases in which there is more than one cerebral lesion), Dr. Gubler meets with embarrassing cases, which he tries to explain by hypotheses which cannot be demonstrated. Such, for instance, are Cases XII. and XVI. of his second memoir. In both of these the paralysis involved the right side of the face and the left limbs, and had set in after a ligature had been put round the right common carotid artery. The first of these cases was published by Professor Sédillot, of Strasburg, in the "Gazette Médicale de Paris" for September 3, 1842. A post-mortem examination disclosed softening of the right hemisphere of the brain, while the pons is not mentioned. Dr. Gubler analyses these cases, and justly rejects the explanation of the facial paralysis given by Professor Sédillot—namely, that it was owing to the facial nerves supplying directly the side of the face corresponding to that of their origin. Although the decussation of the nerves of the seventh pair is not regarded as proved by all anatomists, since M. Sappey has never been able to see it, in spite of the most minute dissections, the fact is admitted, and has been ascertained by Professor Jobert (de Lamballe), by Messrs. Vulpian and Philippeaux, and by Stilling, although the last three state that the decussation

[1] "De l'Hémiplégie alterne envisagée comme signe de lésion de la protubérance annulaire," &c. (Gaz. Hebd. de Méd. et de Chir. Paris, 1856), et "Mémoire sur les Hémiplégies alternes" (in idem, 1859).

is not complete. Besides, the fact that in most cases, paralysis of cerebral origin affects the face and limbs on the same side, tends to prove the existence of a decussation. But if Professor Sédillot's interpretation be faulty, Dr. Gubler's may also raise objections. The arrest of the flow of blood consequent on ligaturing the common carotid is not, to my mind, sufficient to explain, as my colleague thinks, the impairment of motion and sensation which occurred in the corresponding side of the face. No one surely denies the existence of paralysis, or rather of varieties of paralysis, due to an arrest of the arterial or venous circulation; but such paralysis is seen in the limbs only, not in the face, in which there are large and numerous anastomoses between the divisions of the two carotid arteries, which easily permit of a supplementary circulation.

The second case is that of an individual in whom both common carotid arteries were ligatured successively, at an interval of twenty-eight years, for a circoid aneurism of the head. The first ligature was placed round the right common carotid by Dupuytren, and no accident followed; the second time, the left artery was tied by M. Robert. "The result of the operation was as satisfactory as possible; there was some mental excitement only, and the patient insisting on returning home, he had to be discharged two or three days afterwards. The joy he felt at finding himself again among his friends produced still greater exaltation, and brought on delirium, which was soon followed by well-characterized paralysis of the right half of the face and the left side of the body. Death took place shortly afterwards, and a post-mortem examination could not be made."

In this case Dr. Gubler does not explain the facial paralysis by the defective circulation resulting from the obliteration of the artery, as the paralysis involved the opposite side of the face to that on which the artery was tied; but in order to make the facts fit in with his theory, he says: "After the right common carotid had been tied, the circulation of the blood was re-established in the corresponding hemisphere, through the carotid of the opposite side, by means of the communicating artery of Willis, and through the vertebral of the same side, which necessarily increased in size from the innominate retaining its capacity and from the expansive force of the blood-current, which no longer found a wide passage through the carotid, tending of necessity to dilate the vertebral and the subclavian. Now, it *may be presumed* that the vertebral could not be thus distended without its walls being at the same time altered, or the walls of the basilar which is its continuation. This is *all the more probable* that aneurismal dilatation, or at least atheromatous and calcareous changes of the coats of these vessels, are of more frequent occurrence than of other intracranial arteries. The nutrition

of the substance of the brain had, *perhaps*, also undergone some modification, which rendered the organ more liable to become the seat of hæmorrhage. The left carotid being tied under these circumstances, the blood can only flow through the two vertebral arteries, the walls of which are therefore subjected to a relatively enormous pressure. The left vessel, which has healthy coats, resists successfully; whereas the right ruptures, the rupture involving either the trunk of the artery, or one of its branches, on the surface or in the substance of the right half of the pons near the medulla oblongata. The inevitable result of this, in our opinion, is paralysis of the right half of the face, and of the upper and lower limbs on the left side." You see, gentlemen, that however ingenious these explanations may be, they are merely conjectures; and that, instead of drawing his conclusions from observation, Dr. Gubler makes observation fit in with his views.

Of course those cases alone are in question in which the cross-paralysis of the face and limbs is due to one lesion only, for it is conceivable (and Dr. Gubler has called attention to the fact) that cross-paralysis may be caused by several lesions affecting different parts of the brain—a hemisphere on one side, and the facial nerve on the opposite side. But Dr. Gubler does not apply the term "cross-paralysis" to such cases, and confines it to those in which there is a single lesion. But although I do justice to my colleague's essay, and acknowledge that science is indebted to him for having been the first to call attention to interesting facts, and although I admit also that cross-paralysis is often caused by a lesion of the pons varolii, as the cases which he has published show, I think that it is carrying generalization too far, when this form of hemiplegia is regarded as an *absolute* sign of a lesion of the pons. The explanation of this singular form of paralysis escapes us in some cases, and, after all, the same thing happens in a good many cerebral diseases which are still so very obscure.

LECTURE XI.

INFANTILE CONVULSIONS.

The organic alterations are an effect, and not the cause, of the convulsions.—Yet those secondary anatomical lesions should be taken into consideration. Predisposing, hereditary, and acquired causes.—Exciting causes.—The convulsive paroxysm comprises two stages, one of tonic contraction, and the other of clonic movements.—A third stage, that of collapsus, is an effect of the convulsion itself.—Convulsions present infinite varieties.—General convulsions.—Partial convulsions.—Status convulsivus.—Inward convulsions.—Thymic asthma.—Sequelæ.—When death occurs, it is by asphyxia, or by nervous syncope.—Prognosis.—Treatment.

Gentlemen,—Scarcely have a few among you had an opportunity of seeing a baby, who was admitted into the St. Bernard Ward the day before yesterday, and who died the same evening. He was nineteen months old, and had only cut six teeth. He had, for the last few days, been seized with convulsions, recurring in paroxysms four or five times in the twenty-four hours. About a year previously, when cutting his first teeth, he had been seized in the same way, and the attack had lasted eight days, as on this occasion, but the symptoms had been different from those witnessed this time, and had constituted what are termed *inward convulsions.*

When his mother brought him to the Hôtel-Dieu, the child had therefore been ill for eight days. Yet, he had not been convulsed on Sunday last, and he seemed well, when the convulsions returned on the following day with renewed intensity, so much so, that since Tuesday evening (he was admitted on the following Thursday) they recurred almost uninterruptedly. Since that time also, he refused the breast, and remained in a condition of true status convulsivus.

The convulsions returned every four or five minutes, each paroxysm lasting from thirty-five to eighty seconds. Although they were very rapid, I could still observe that they consisted of two very distinct periods; namely, first, a stage of tonic convulsions, succeeded by clonic ones, which, in the interval of the paroxysms, still persisted in a certain degree, and were exaggerated on the recurrence of the fit. The arms and legs executed extensive movements through the involuntary contraction and alternate relaxation of their muscles. From the commencement of the paroxysm, there was convergent strabismus, and the eyes

looked down towards the lower eyelid; the urine also was passed involuntarily.

There was febrile reaction, shown by heat of skin, and acceleration of the pulse (168 in the minute), and the child's mother stated that he had been feverish from the beginning. Lastly, the child coughed, but on carefully examining his chest, nothing abnormal was detected.

The autopsy disclosed no lesion of the nervous centres; yet it seemed to me that the grey matter of the cerebral convolutions was of a slightly deeper tint than it normally is. The lungs were slightly congested and emphysematous, especially the middle lobe of the right lung.

You will be frequently called upon in the course, and even at the outset, of your medical career to attend cases of infantile convulsions, and the subject is of such great importance that I mean to devote a few conferences to it.

Convulsions, considered generally—and I need not insist on the fact—are met with in a good many morbid conditions of very different nature. In some cases, they seem to arise from manifest anatomical lesions of the nervous system; in others, they seem to be caused by no material change, or, at least, the most rigorous examination after death reveals the existence of no organic alteration to which may be ascribed the morbid phenomena which manifested themselves during life. Hence a great primary distinction between so-called *symptomatic* and *idiopathic convulsions*.

These latter may be the expression, and sometimes the sole expression, of very different diseases. I have shown you how of themselves they characterized one form of epilepsy, the *haut mal*; and you know well the important part which they play in hysteria. They again constitute the predominating symptoms in the various forms of chorea; and the permanent involuntary muscular contractions of tetanus, and of the affection described under the name of idiopathic contraction, are only tonic convulsions. Lastly, under the generic term, idiopathic convulsions, are included the various forms of *eclampsia*, to which *infantile convulsions* should be referred.

One point is first to be elucidated, before I enter upon their clinical study. I stated that idiopathic convulsions could not be ascribed to the presence of any appreciable anatomical change, but I did not mean thereby that they were independent of a material affection undoubtedly seated in the nervous centres; I merely assert that the most minute dissections have not yet taught us (if they ever can succeed in so doing) the organic pathological condition in consequence of which convulsions arise.

I still less deny that dissection discloses in individuals, who have died in convulsions, more or less extensive lesions of the

nervous system, but I will repeat what I have said already when treating of epilepsy, and what I will say of all neuroses, that those lesions are but of secondary importance in the history of the disease. They are, for the most part, the result of disturbances of the nervous system, perhaps of those inappreciable organic modifications to which I have alluded, which have taken place in the nervous system, but they are conse quences and not a starting-point.

Thus, a child is seized with convulsions and dies. On dissection, a more or less marked congestion of the meninges, the brain, and spinal cord, and serous effusion into the ventricles or into the arachnoid sac, sometimes even one or more hæmorrhagic centres, are met with. Now, are the congestion and the effusion to be regarded as the causes of the convulsions? Surely not. They no more caused them than the cerebral congestions and hæmorrhages which take place in epileptic fits are the cause of the fits; no more than the pulmonary engorgement and serous effusions into the pleural cavities, sequential to paroxysms of asthma, caused the asthma. What we see is the analogue of that transient congestion which brings colour into the cheeks of an individual who is under the influence of anger or of a deep mental emotion, and which is, in some cases, carried to such a degree as to involve the brain itself. It may be also compared to the congestions which accompany neuralgic affections, phenomena to which I call your attention every day, and which have been described by Dr. Notta in an excellent Memoir.[1]

The opinion which I maintain, and which is accepted by most practitioners, is far from being novel; it was very clearly professed by Morgagni when he wrote in his eighth letter "*De causis et sedibus Morborum*:" "The cause of convulsions, which consists in an invisible change that has occurred in the brain and nerves, cannot be detected by our senses after death; its effects alone are seen, and these vary according to the violence and duration of the convulsions." Yet, gentlemen, do not go beyond my meaning, and believe that I attach no importance to these material lesions. Although they occupy but a secondary place as the effects, and not the causes, of convulsions, they should not the less be taken into consideration. For, if when slight, they disappear rapidly and spontaneously as soon as the cause which produced them ceases to act, they are capable, when carried to a very high degree, of bringing on the most serious complications. When they recur frequently, they may cause anatomical changes, and subsequently incurable functional disorders; if there be no immediate danger of life, the patient

[1] Mémoire sur les lésions fonctionnelles sous la dépendance des Névralgies. 'Archives gén. de Médecine,' 5e série, tome iv. juillet, septembre, novembre 1854.

becomes, at least, subject to incurable infirmities, as we have seen; for instance, epileptics remain paralysed after convulsive seizures; *à fortiori*, should we take into account the extravasations of blood which result from an attack of eclampsia. I shall revert to those points.

We cannot find out, then, what the ancients called the proximate cause of convulsions, but we know better their *predisposing* and *exciting causes*.

In our conferences on epilepsy, I tried to prove to you by facts the influence of *hereditary predisposition* on convulsive disorders. This nervous susceptibility manifests itself in different generations either in the same or in a different way. It pretty commonly happens that parents, mothers especially, who in their infancy were subject to fits, give birth to individuals who are in their turn affected in the same way.

One of the most extraordinary instances of the kind, which I know of, is that related by my old pupil and friend, Dr. Duclos (of Tours) in his remarkable thesis.[1] The case is that of a woman, thirty-four years of age, the sister of ten children, six of whom died of convulsions, and who had herself had frequent attacks of eclampsia up to the age of seven. These had left behind slight deviation of the mouth and ptosis of the left upper eyelid. This woman had ten children, who all had convulsions; six had died, five in the first two years, and one when three years old. Her youngest, whom she brought to me at the Necker Hospital, was a little girl six months old. Three months previously she had had a first attack, which had lasted about ten minutes, and which her mother ascribed to her having given the breast to the child immediately after a fit of passion, as the convulsions occurred on the ensuing day. Death took place, three months afterwards, from cerebro-meningitis.

Accoucheurs have often remarked that infants, whose mothers had eclamptic seizures shortly before delivery, were liable to convulsions within a short time after birth. In some cases, death results from the violence of the fit; in others, the child gets well although the paroxysms have been very frequent and have recurred at very short intervals. The same authorities state also that they have seen infants at birth with contractions of the limbs or muscles of the neck, which were the result, according to them, of convulsions or of some analogous affection at least from which they had suffered *in utero*, the mothers having had convulsions during pregnancy.

Independently of this predisposition transmitted from parent to offspring, there are a series of causes which predispose to

[1] Études cliniques pour servir à l'histoire des convulsions de l'enfance. Paris, 1847, p. 75.

convulsions in a singular manner, namely, all those which tend to weaken the system. Hence convulsions are most frequent in children who are *insufficiently fed*, who have lost a relatively large quantity of blood, whether from spontaneous hæmorrhage, or from venesection, or the application of leeches. Profuse *diarrhœa*, persisting for a long time, acts in the same way. This need not surprise, if this great physiological law be kept in mind, namely, that in proportion as the nutritive and vegetative functions are feeble and languishing, nervous phenomena are mobile, exalted, and irregular—a law which has been admirably enunciated in this simple observation of Hippocrates, *sanguis moderator nervorum*; if it be especially remembered that the dependence of the nervous system on the blood and the nutritive functions is most strikingly marked in children.

I shall not enumerate to you the long list, given by authors, of the exciting causes of infantile convulsions. I shall only remind you that a high temperature, sudden exposure to cold, mental emotions, and *local irritation*, can bring them on.

A few years ago, I was asked to see, with my friend and colleague Dr. Blache, the child of a foreign minister accredited to the French government. The child had for some hours been seized with paroxysms of convulsions, for which he had been put in a bath. The convulsions did not cease, when Dr. Blache, on removing the child's cap, saw a piece of thread across his head, and on trying to take it away, pulled out a long needle which had entered the brain. The convulsions ceased immediately, but hydrocephalus set in shortly afterwards and carried off the patient.

A son of my excellent colleague Professor Soubeiran, having died of convulsions, for which no cause could be assigned, a *post-mortem* examination was made, when a needle was found transfixing the liver, and to this cause the convulsions were referred.

Underwood relates a case like my first in his Treatise on *Diseases of Children*.

A child, after incessant crying, had been seized with convulsions which could not be clearly accounted for by the medical attendant until after death. On removing the child's cap, a small pin was found sticking into the anterior fontanelle.

Bear these facts in mind, because you may happen to see convulsions cease when you find, on undressing the little patient, that a badly-placed pin, or even a painful constriction of the dress, was the starting-point of the convulsions.

Remember, also, that fits are often brought on by the application of blisters, of sinapisms to the limbs of children, with the intention of combating nervous disorders of no gravity. How often have I seen convulsions which terminated in death,

supervene in children that had been covered over with blisters; and how often have I seen medical men use fresh blisters against the evil they had themselves caused, forgetting the nervous symptoms which so frequently accompany a burn of the first degree.

The convulsive seizures that are so common in some children, not only during the first dentition, but also, although much more rarely, during the second, are to be ascribed in a great part to the irritation caused by the difficult evolution of the teeth.

In an etiological point of view, convulsions that are connected with well-defined physiological conditions, are undoubtedly the most interesting to study. Those which are due to an appreciable organic alteration of the nervous centres, such as the convulsions of cerebro-meningitis, need not occupy our attention, and the history of such *symptomatic convulsions* forms part of that of the disease of which they are one of the manifestations. But *convulsions* which, from their occurring at the onset, during the course or towards the close of various diseases, are termed *secondary*, and said to result from *sympathy*, are referable to eclampsia properly so called, of which those diseases should be regarded as exciting causes. Such are the convulsions which occur at the outset of eruptive fevers, of measles, and of small-pox, more frequently than of scarlatina; at the commencement of pulmonary or intestinal catarrhal affections; in a word, of most of the inflammations or fevers which attack children.

Apart from these catarrhal or purely inflammatory affections, and from chronic diarrhœa, *disorders of the alimentary canal* have the greatest influence on the production of convulsions.

Indigestion is one of their most frequent causes, whether due to excess in the quantity of food, as when the child is given too much milk which is good in all respects; or whether it is the consequence of the use of coarse food, which is not adapted to the age, the digestive capabilities, and the individual dispositions of the child, as when infants at the breast are fed, at too early an age, on thick panadas, on haricots, on lentils, or on potatoes, &c., as you will too often have occasion to see.

I wish to dwell very strongly on a point to which I have already called your attention, and to which I cannot too often revert; namely, that, contrary to the generally accepted notion, children at the breast, who are subject to diarrhœa, are much more frequently liable to convulsions than those whose motions are habitually regular, not because diarrhœa predisposes to eclampsia more particularly, but because persons whose bowels are delicate and often disordered, are more than others liable to indigestion, which is a powerful cause of convulsions. Hence

I have for many years laid down a rule for myself, to stop the diarrhœa of children even when they are teething. Cases have been recorded of children who had been seized with convulsions, after taking the breast soon after the mother had felt a violent emotion. I saw at the Necker Hospital, to which I was physician for a long time, eclampsia come on in a child whose nurse had had a violent fit of passion a moment before giving him the breast. Professor Andral related in his lectures still more curious instances, showing that there are singular idiosyncrasies, under the influence of which the milk of a nurse is well digested by some children and not by others. "A woman nursed her own child without any ill effect, but another child to whom she gave the breast was seized with convulsions, and a third likewise." In all such cases, the seizure comes on without any other symptom of indigestion being present; it seems as if the nature of the milk was altered under the influence of some cause or other, and it became a poison acting on the nervous system.

I have already called your attention to the important fact, pointed out for some years past, of eclampsia coming on in children as well as adults, as a consequence of albuminuria, whether it occurs in the course of an acute affection, as it often does at the close of scarlatina, or whether it be a symptom of Bright's disease.

In such cases, the patient has generally had more or less considerable anasarca; but you must not think, as some seem to admit, that anasarca is the most favourable condition for the development of convulsions; because, on the one hand, children who become anasarcous without passing albuminous urine after an attack of dysentery, of obstinate diarrhœa, of measles even, are rarely seized with eclampsia, whilst in albuminuria without anasarca, convulsions are of frequent occurrence, and so much so, that some authors have not hesitated to affirm that, in nearly every case, infantile convulsions were a symptom of albuminuria, an opinion which to me seems exaggerated. Those authors have even tried to diagnose eclampsia from epilepsy by the presence of albumen in the urine in convulsions. I have often reminded you of Professor Claude Bernard's curious experiments on the influence of injuries to the fourth ventricle on the urine. If, in an animal, this ventricle be injured in a certain spot, the urine is found to contain sugar within a short time, and to be secreted more abundantly. If some other spot be wounded, mere polyuria follows, and no sugar is found in the urine. An injury to a third spot soon renders the urine albuminous.[1]

[1] Leçons de Physiologie expérimentale appliquée à la Médecine faites au Collége de France.

Can it be supposed, then, that the same venous modification which, in a child or in a woman, produces albuminuria, causes a liability to eclampsia?

The presence of *worms* in the alimentary canal has been mentioned by all authors as one of the most common exciting causes of infantile convulsions, and I have already told you of a case of epilepsy which was cured by the expulsion of a tænia. Such cases are instances of *reflex convulsions.*

Without attempting to review all the causes of infantile convulsions, I will call your attention to this peculiarity; namely, that circumstances apparently the most insignificant may bring them on in individuals predisposed to them; that there are children who are convulsed with as much facility as others pass into a dreamy or delirious state; and that this predisposition is chiefly hereditary. As I have already told you when speaking of epilepsy, this nervous susceptibility or excitability may in some cases be foreseen. Care, however, should be taken not to mistake for convulsions the rapid and *involuntary* movements which occur, even in the waking state, in individuals whose nervous system is very excitable, when surprised by an unexpected noise, or when under the influence of sudden mental emotion. Such movements are convulsive in appearance only, but fail to present the essential characters of convulsions properly so called.

Let us, then, see what these characters are. Viewed in its simplest element, a convulsive seizure consists of two successive and very distinct stages. The first stage is one of contraction without shocks, consisting in a gradual but rapid shortening of the muscular fibres, shown by the hardness and stiffness of the affected muscle, which cannot be overcome in some cases. This *period of tonicity* is soon followed by a *clonic stage*, characterized by the occurrence of alternating movements of contraction and relaxation independent of the will, which is as powerless to suspend or moderate them as it was to excite them.

The tonic always precedes the clonic stage, but the duration and violence of the latter are by no means proportionate to the duration and violence of the former. Thus very violent clonic movements often succeed a slight tonic contraction, and reciprocally an excessively powerful tonic contraction may be succeeded by very moderate clonic movements. Thus the length of the first stage is sometimes so short, and the second stage comes on so quickly, lasting for a more or less prolonged period, that an observer who is not on his guard, or not very attentive, might think that the convulsions were clonic from the outset. In other cases, which are not so frequent it is true, there are no clonic convulsions, and there is only during the whole time a muscular contraction more or less energetic and persisting.

This is what occurs in idiopathic contractions, of which I shall speak on some other occasion. In eclampsia, more especially in the eclampsia of children, of which alone I mean to treat to-day, clonic convulsions are absent when death occurs during the fit, and as a consequence of the length of the tonic contraction, from asphyxia or syncope, by a mechanism which we shall investigate by-and-by.

From what I have said, this remarkable fact follows, that rigidity seems to be an essential, obligatory element of all convulsion. It is never absent, and can even be alone present, whether it constitutes the convulsion by itself, as in idiopathic contractions, or whether the convulsion is incomplete, as in eclampsia, when the clonic stage is absent, whereas clonic movements never perhaps come on from the first.

There is a third stage which should not be omitted, although it does not form part of the convulsive seizure, namely, that of *collapse*, *stupor*, or *coma*. Whether it be the consequence of cerebral surprise, produced by congestion or exhaustion of the nervous excitability, this collapse is an effect, and not the cause, of the convulsions. If in most cases there sets in after an attack of eclampsia, more or less profound, or more or less transient stupor, in some instances of very infrequent occurrence, it is true, there is no transition between the attack and the return to a normal state.

After this analysis of the various phenomena which constitute a convulsive fit, I now pass on to a more general study of infantile convulsions, and will attempt to describe them as completely as possible. The subject is one of extreme difficulty, for eclampsia assumes the most varied forms. More commonly it comes on like an epileptic fit. Nothing foretells the invasion of the attack; and, for my part, I have never observed the premonitory signs spoken of by Brachet, and repeated after him by others. The state of impatience, of uneasiness, of agitation, and *malaise* that is spoken of, the light sleep or the wakefulness, sometimes, on the contrary, replaced by languor, hebetude, and somnolence, are the prodromata of the disease, of which the convulsions are a primary manifestation, and cannot be referred to the convulsions themselves.

The convulsions set in suddenly. The child utters a cry, loses consciousness, becomes rigid, and struggles, with a fixed chest and suspended respiration; the face, which is pale at first, becomes red and livid; the eyes sometimes fill with tears, which run over the cheeks; the veins of the neck project like knotted cords. The clonic stage then begins, characterized by disorderly and involuntary contractions of a great many muscles; the limbs are alternately flexed and extended; the fingers and toes are successively bent and stretched out, separated from or approximated

to one another, but most frequently they are in a state of forcible flexion; the thumb is adducted and hidden by the fingers. The head is drawn backwards or is bent forwards; sometimes it is pulled laterally by irregular and jerking rotatory movements; the muscles of the face share in the *general convulsions*; the eyes are the seat of jerking movements, and roll in their sockets; they are generally drawn up under the upper eyelid; more rarely they are pulled downwards, and there is strabismus convergens. The labial commissures are dragged upwards and outwards, and hence the distorted face is sometimes frightful to behold; then, on each convulsive shock, the air passes through the kind of funnel formed by the corners of the half-opened buccal orifice, and makes a suction noise, accompanied by a flow of frothy and sometimes bloody saliva. In this, as in an epileptic fit, the tongue is protruded, and may be bitten and lacerated by the teeth. As the muscles of the trunk are likewise affected during the tonic stage, the inspiratory muscles are fixed, and the larynx itself, which is spasmodically contracted, no longer admits of the free passage of air. The abdominal muscles being thrown into convulsion, cause the involuntary expulsion of the urine and fæces. The clonic convulsions, at first rapid and limited, become slower and more extensive, and at last a deep inspiration, followed by complete relaxation, announces the end of the fit. The child then falls into a state of somnolency and stupor.

These various phenomena take place in much less time than I have been in describing them, and the fit, which is always too long for the frightened mother, lasts one or two minutes. When it is completely over, and when, after the stupor has disappeared, order is re-established, it is impossible to see any traces of what has passed, beyond that the child shows signs of fatigue by yawning and by a tendency to sleep.

The paroxysm may consist of a single fit, but this is rare. Generally, after a more or less prolonged pause, a second fit comes on, having the same characters as the first, lasts the same length of time, and is, like it, succeeded by coma, after which the child recovers his normal condition.

Like the first, it comes on without any appreciable cause, but it may be brought on by emotion, by annoyance, by pain, or by movement, and may recur every hour, every half-hour, and even at nearer intervals.

An attack of eclampsia thus composed of several fits, may last from half a day to one, two, or three days; and there are instances in which it has been prolonged beyond that time, the child being liable to convulsions, which, recurring at more or less short intervals in the course of the twenty-four hours, are continued over five, six, seven, and fifteen days, as in cases

reported by Dr. Duclos,[1] and even over eighteen days, as in the case of a child, five months old, whom I saw at the Necker Hospital.

During an attack of whooping-cough, this child had every day one or two paroxysms, consisting of a series of subintrant fits; that is to say, of fits following one another so quickly that one fit was not yet over before the next one began; the paroxysm lasted in this way, without the least interruption, for two, three, and even four hours.[2]

This, gentlemen, confirms what I told you just now as to the variety of forms assumed by infantile convulsions.

Generally *intermittent*, separated by intervals of rest, during which order seems to be re-established, they are found, at other times, as in the above case, to succeed one another without intermission. A fit which has lasted one or two minutes, is scarcely over when it is followed by another, which is, in its turn, succeeded by from one to twenty other fits; so that the little patient merely passes from the contortions of convulsion into a still more awful torpor; and from want of attention, many medical men consider this state as one of continued convulsion.

A little care is alone needed in order to recognize a series of paroxysms, the violence of which is generally, in such cases, less great than that of intermittent convulsions. This condition resembles exactly what in epilepsy I termed the *status epilepticus*, a condition of much less frequent occurrence, however, in epilepsy than in eclampsia.

These apparently continuous convulsions may last a considerable length of time, for eight or ten hours, then, after a more or less prolonged interval, may again assume a continuous form, and recur from one to two and even to fifteen or seventeen days in succession.

This form of eclampsia, therefore, differs from the one in which the attacks are markedly intermittent, only as regards the mode of recurrence of the fits.

There is, however, a *continuous form*, which is pretty often met with after a violent epileptiform seizure. Just as the jerks are expected to cease, they return every second or at slightly longer intervals, and this goes on for a quarter of an hour, for an hour, or even for whole days. In such cases, there is, in reality, but a single attack; for although from time to time the convulsions seem to diminish in intensity to begin again with renewed violence, there is never a complete cessation of the convulsions, nor the profound stupor and the general muscular relaxation which follows on an ordinary paroxysm. In this

[1] *Loc. cit.* p. 23.

[2] Id. p. 23.

continuous form of eclampsia there is a capital symptom to which I wish to direct your attention at once. Whereas in the first two forms, there was loss of consciousness, in this form, on the contrary, although the child is convulsed, he seems as if he had not entirely lost consciousness, and was not a complete stranger to all that was passing around him. He expresses his wants or pain by cries; he occasionally withdraws with a certain amount of vivacity his hand when it is pinched, or his foot when it is tickled, although the very limb is convulsed which still responds to the commands of the will.

In truth such convulsions, which affect the whole body, are not so *general* as they seem to be, since there are some muscles which still obey the will, and they must therefore be regarded as *partial*, strictly speaking.

Let us now pass on to more localized convulsions. *Partial convulsions* present the most marked differences, and their infinite diversity of form is in direct relation to their seat. It sometimes happens after an epileptic fit that *one-half of the body* is for several hours the seat of spasmodic clonic movements, which recur at intervals of from one to several seconds. The child is yet perfectly conscious, and the movements of the opposite half of the body are executed with an ease and a co-ordination which contrast singularly with the agitation of the affected side.

I remember seeing a little boy of eleven months old with the tubercular diathesis, who, after a violent attack of eclampsia, recovered his senses, but whose right arm and the right half of whose face were convulsively and violently agitated for several hours. He knew his mother and his nurse; could drink, although with some difficulty; looked with attention and intelligence at all the objects around him, turned his head quickly round to look at the persons who entered the room, and sometimes even, worried by the jerks of his right arm, tried, by holding it with his left hand, to stop the violence of the convulsive movements.

In other cases, instead of affecting in an equal degree all the muscles on one side of the body, convulsions affect unequally one muscle or another, and often muscles which are not supplied by the same nerve. Thus only one muscle of the arm, the biceps, for instance, may be convulsed, while the others are perfectly quiet and relaxed, and one or more fingers are alone moved. Occasionally, although more exceptionally, the lower limbs are the seat of these partial convulsions.

They are frequently preceded and accompanied by the general disorders which characterize an epileptic fit, such as a scream, loss of consciousness, and pallor of the face; often, also, the attack is followed by a period of stupor and carus. These

phenomena are never more marked than in the cases when the convulsions are limited to the trunk, cases, which although not rare, are yet much less frequent than those in which the limbs are exclusively convulsed.

Partial convulsions of the trunk assume, besides, two very distinct forms. They are sometimes incomplete, and consist of an exclusively tonic contraction of the muscles of the vertebral groove, which resembles a true tetanic spasm. The body stiffens; the head is drawn backwards and immovably fixed; and then, without there having been any flexion, all contraction ceases, and the normal condition is restored. Sometimes this tonic stage is so transitory that it seems to be absent, and clonic contractions are alone seen, which make the head rotate or bend forwards or backwards, the convulsions seeming to be exclusively limited to the muscles of the neck. I will here repeat an observation which I have made already, namely, that you should not confound with convulsions certain movements which somewhat resemble them, and which recur in a great many children during a febrile affection. Such movements show the excitability of the little patient, and although they are due to an exaltation of the nervous system, are in reality not convulsive.

Of all partial convulsions the most frequent are unquestionably *those of the face.* They sometimes involve all the muscles of one half of the face, when the eyelids, the globe of the eye, the ala nasi, the cheek, are thrown into convulsive contractions, the mouth is distorted, the lower jaw is depressed and pulled to the affected side, and the teeth are set, or there is a kind of chewing movement continually going on. In some cases the convulsions are still more limited in their area, involving either the orbicularis palpebrarum, and causing rapid involuntary winking, which lasts more or less time, or a few muscular bundles of the cheeks and lips, the commissure of the latter being then violently dragged upwards and outwards, or the muscles of the ala nasi, causing alternate dilatation and closure of the nostril. The muscles of the *tongue* are sometimes affected, and articulation being then impossible, there results a kind of stammering, which is generally transient, but sometimes persistent.

Convulsions of the muscles of the eye are the most frequent of the partial convulsions of the face, and I will go so far as to add that they are perhaps very often overlooked. They generally announce the invasion of an attack of eclampsia, but sometimes also they are the only symptom of the complaint. They are, in some cases, exclusively tonic; the globe of the eye is drawn up and hidden under the upper lid, or there is double and convergent strabismus; in exceptional cases the strabismus is divergent. In other instances one eye is alone affected, and the other is perfectly motionless; the strabismus is then almost

always convergent. It may also happen that the strabismus is convergent on one side and divergent on the other. Usually the convulsions of the muscles of the eye are complete; that is to say, permanent contraction is succeeded by clonic movements, and the globes of the eye oscillate continuously, being drawn up under the upper lid, and then pulled down under the lower one, and looking inwards towards the nose much more frequently than outwards.

With respect to one point you must be on your guard. You will probably be more than once called upon to see children said to have been seized with convulsions, because their eyes shall have been seen to be drawn up under the upper eyelids whilst they are asleep, and this essentially physiological condition has been mistaken for the consequence of convulsions. The eyes are sometimes drawn up to such a degree that on separating the lids, the iris, and the pupil particularly, cannot be seen at all. The pupil is besides completely contracted, whereas during convulsions it is, on the contrary, more or less dilated. This physiological condition gives rise to frequent mistakes, especially when children have recently had true convulsive seizures.

It is easy, gentlemen, to recognize eclampsia under the different forms which I have just reviewed, however elementary or partial it may be. These forms are perfectly distinct and special, and all medical men agree, if not on the nature of the disease, at least on the name which should be applied to it. The case is always one of *convulsions*. But opinions have differed, and still vary very much, with regard to the forms to which I now pass on, namely, *inward convulsions*, to which, when slight, some authors have applied the term *spasms*; and which, according to the muscles that are affected, give rise to phenomena which are differently and sometimes singularly interpreted.

By inward convulsions are commonly meant partial convulsions, more particularly of the pharynx, of the larynx, and of the whole muscular respiratory apparatus. The term has certainly not a very clear and precise meaning, but it is good enough, provided its meaning be well understood.

The most common form of inward convulsions consists in the drawing up and the mobility of the globe of the eye, of which I spoke just now; in a nearly complete loss of consciousness, or at the very least in pretty profound stupor; in extreme difficulty or inability to swallow; in uneven breathing, sometimes scarcely perceptible, sometimes deep, broad, and blowing, showing that the diaphragm and the respiratory muscles of the abdomen and chest are more particularly involved; sometimes there is heard for one or several minutes a peculiar laryngeal whistling, which indicates an obstacle to the entry and exit of the air, a circumstance to which I shall revert presently.

Inward convulsions may coexist with general or partial convulsions of the limbs and face (for I have told you that these were usually accompanied by convulsions of the globes of the eyes), or they may be alone present. In either case again, they may be complete, that is, tonic and clonic, or incomplete, and consequently consisting in tonic contraction alone. If, in the former case, the patient be exposed, the *convulsions of the diaphragm and the respiratory muscles* may be seen to cause very rapid and frequent, though not extensive, movements of the base of the chest; in the latter case, the base of the chest is violently drawn in and remains immovable. Clonic convulsions, owing to the frequency of their recurrence and the shallowness of their movements, necessarily produce profound perturbation of the respiratory function, which becomes embarrassed, and therefore of hæmatosis. Again, the convulsive jerks explain the slight and peculiar fits of coughing which frequently accompany inward convulsions.

Tonic convulsions suddenly arrest and completely suspend the respiratory functions. Hence you can easily understand that they cannot last for a long time continuously, without causing death. So that, whereas convulsions of the limbs and face may extend, without any inconvenience, over a minute and a half and two minutes; tonic convulsions of the diaphragm and of the inspiratory muscles must be transitory only, and cannot last over a minute without immediate danger.

Inward convulsions chiefly consist, then, in convulsions of the diaphragm and of the respiratory muscles of the abdomen and chest; but it happens also that the intrinsic muscles of the larynx are convulsed simultaneously, and this *laryngeal convulsion* causes again disorders of respiration, which may, in some cases, excite serious alarm. A rickety child, subject to epileptiform convulsions, which, for the last few months, had recurred several times a day under the influence of the least fit of anger, was one day brought to me at the Necker Hospital. He had besides, from time to time, attacks which his mother could not describe clearly, but which, according to her statement, were still more grave than the great seizures. Several of these attacks occurred in my presence. The child suddenly threw himself backwards, his throat was tense, his mouth half-opened, his eyes fixed, his arms and legs moved by convulsive jerks. Quick inspiratory movements created inside the chest a vacuum, which was immediately removed by the falling in of the ribs; air seemed not to enter the larynx, or if a little went in, it caused a sharp whistling noise, somewhat similar to that which is sometimes heard during the most violent paroxysms of croupal dyspnœa. During the attack, the face, neck, and chest, and the mucous membrane of the mouth became more and more

livid in hue, until, as the spasm ceased, one or more deep inspirations put an end to this terrible scene. Profound depression, like what follows an attack of eclampsia, then ensued. It is these convulsions which affect the respiratory apparatus, and the larynx more particularly, that constitute the disease described by Kopp under the name of *thymic asthma*, the *laryngismus stridulus* of Hood and of Ley, and on which my colleague Dr. Hérard has written a good monograph.[1]

Allow me, gentlemen, to dwell a moment on this question, which has given rise to much discussion. You are aware that this complaint has been ascribed to an abnormal development of the thymus gland, but it has been conclusively shown that it is perfectly independent of it. First of all, is it necessary to state that the thymus gland, as well as the suprarenal capsules, organs of transition which are destined to atrophy after birth, are, less than other organs in the body, placed in conditions that give rise to hypertrophy? For more than twenty years I was attached to a hospital where a large number of very young children was admitted, and I *never once* saw swelling of the thymus gland that was capable of giving rise to the slightest accident. Besides, is it conceivable that the thymus may grow to such a size as to obliterate the trachea to a great degree without the child's friends and the medical attendant being warned by the presence of habitual dyspnœa? And if there has never been any dyspnœa, can one understand by what process an organ, which contains so few bloodvessels, may in a few minutes become a cause of death, or, at least, of awfully serious accidents? Now, if the hypothesis be suggested, that the gland, on undergoing hypertrophy or alteration, has involved the recurrent laryngeal nerve, as in cases of tubercular infiltration of the lymphatic glands of the neck and the roots of the bronchi, how can one believe that there has been no modification of the voice or of respiration, and that the disease reveals itself only by a sudden attack of dyspnœa?

Pathological anatomy has by this time thrown sufficient light on this contested point, and has shown that if the thymus is sometimes abnormally developed, its hypertrophy is not necessarily attended during the child's life with symptoms of the so-called thymic asthma, whereas in fatal cases of inward convulsions like those described under the name of thymic asthma, the gland has undergone no alteration. The study of the symptoms could, after all, but lead to the conclusion that convulsions were alone in question, for on investigating the series of the forms of eclampsia, one can easily recognize convulsions affecting the respiratory apparatus, the diaphragm, and more particularly the larynx.

[1] Du spasme de la glotte (Thèses de Paris, 1847).

Who does not see that there need only be a want of harmony between the spasmodic movements of the diaphragm and those of the muscles which move the arytenoid cartilages, in order to give rise to the laryngeal whistling and the dyspnœa? In a regular act of inspiration, the upper portion of the larynx opens, while the diaphragm is depressed and makes a vacuum inside the chest. Now, if the diaphragm be depressed too rapidly, and there be at the same time laryngeal spasm, as happens in hooping-cough, inspiration becomes almost impossible, and is accompanied by a very loud whistling.

In the present instance we need not have recourse to a want of harmony between the movements of the diaphragm and those of the laryngeal muscles; we need only suppose that the will or instinct no longer presides for a moment over the movements of the arytenoid cartilages; the muscles which move these cartilages, no longer responding to a nervous impulse, are for the time in the same condition as those of animals whose recurrent laryngeal nerve has been cut.

What occurs deep in the larynx may sometimes take place under the observer's eye. In order to test the truth of the theory which I had framed to myself regarding the so-called *thymic asthma*, I have on occasions remained a long time by the side of a child suffering from convulsions of the diaphragm, without participation of the larynx, and have brought on at will the phenomena of *thymic asthma*, by closing for a moment the child's mouth and nose.

When the mouth was closed, and the nostrils pressed slightly, so as to occlude them for a second only and then to leave them half-opened, at the moment when a greater convulsion of the diaphragm carried the air more rapidly through the nasal fossæ, the alæ nasi, yielding to the pressure of the air, were seen to press against the septum, and so intercept the passage of air, so that immediate suffocation resulted. The reason of this was, that during the convulsions, the alæ nasi did not open during the forcible inspiration, as they do in a physiological and even a pathological condition.

I need not remind you of the distinction between *thymic asthma*, and the *acute asthma* of Millar: the latter is stridulous laryngitis, in which the spasm of the larynx which characterizes it is due to an inflammation of the respiratory tract.

Thymic asthma may be preceded or accompanied by other symptoms of eclampsia, but it may also be the only manifestation of the complaint. It may set in suddenly in the midst of apparently splendid health, without any appreciable cause, but it more commonly comes on under the influence of some mental emotion or of a fright. I was once consulted for a little boy, who from the beginning to the end of his first dentition was

subject to such seizures. He was of a very excitable temperament, and the least annoyance brought on an attack: although he is still very excitable, he never, however, has any such attacks now.

Remember what I told you already in our conferences on epilepsy, that these laryngeal spasms, and eclampsia, in general, are, in some cases, the prelude of epilepsy, which, as the individual grows older, manifests itself more clearly. On this account, therefore, you should be extremely reserved in your prognosis of thymic asthma; and for a greater reason still, namely, that the patient may be carried off in a fit, when it lasts beyond a certain time, although when the attacks are very short, they are not grave in themselves. Indeed, it is very remarkable that eclampsia in children generally leave after it no traces of its passage, even though the seizures have been frequent and violent, and have recurred during five, eight, ten days, and even more. The little girl, whom I spoke of in the course of this lecture, got perfectly well, and her health did not seem to have suffered in the least from the convulsions which had recurred during eighteen days.

In some cases, however, convulsions are followed by *sequelæ*, which may be temporary, or may be persistent and irremediable. Thus, muscles which have been particularly and most violently convulsed, are sometimes, after an attack of eclampsia, the seat of pretty acute pain, resulting either from laceration of their fibres, or effusion of blood, when the pain is not simply a consequence of the fatigue felt after exaggerated muscular efforts.

In other cases, more or less incurable deformities result from attacks of eclampsia. You are aware that among the theories propounded to explain certain distortions of the neck in newborn infants, certain *congenital deformities of the limbs*, and *talipes* in particular, there is one which admits the influence of convulsions of the fœtus *in utero*.

These infirmities may be brought on after birth; and you know that eclampsia is regarded as one of the most frequent causes of *squinting* and of *stammering*. It should be added, it is true, that these acquired infirmities are most frequently the result of convulsions that are symptomatic of an appreciable lesion of the nervous centres, and that they are then less due to the convulsions themselves than to the persistent organic cause which brought on the fit.

Sometimes, again, eclampsia is accompanied or followed by *paralysis*. In some instances, the parts which were convulsed are, after the attack, markedly weak, and this weakness may be carried to absolute loss of motor power; in other instances, the limbs on the opposite side are paralysed; lastly, the upper

limb may be convulsed, while the lower limb on the corresponding side is paralysed. The paralysis is generally transitory, like the convulsions which accompany it; but it may last more or less permanently after the attack. It may be partial also, and, like the convulsions, may affect only one or several muscles. This is especially the case with the face; and these accidents, which occur either on the same side with the convulsions, or on the opposite side, seem to give rise to a certain number of cases of *facial paralysis*, the origin of which is vainly sought for elsewhere.

This secondary paralysis partly explains some of the deformities to which I have alluded, and which are, in fact, owing to the permanent contraction of one or several muscles. Now, if contraction may follow on convulsions, it is well known also that muscles, which have been long paralysed, are, after more or less time, subject to it. Lastly, *idiotcy* very often supervenes on infantile convulsions; and it rarely happens that in such cases one half of the body is not weaker than the other, the paralysed side being less developed than the sound one. It is then probable that the convulsions have been accompanied or followed by deep lesions of the nervous centres.

Although these sequelæ of convulsions are not very rare, on the other hand, they are not frequent in proportion to the extreme frequency of eclampsia; and I may here repeat what I said just now, namely, that, generally speaking, this complaint is usually of no gravity. The fatal cases, however, to which I have more than once alluded, and in which *death* was *an immediate consequence of the attacks*, are still too frequently met with for not warning you of the possibility of this awful termination. It is to be dreaded, not only after numerous attacks, recurring in rapid succession, but even in a first attack. Death then takes place, either by *asphyxia* (and this is the most common mode), or by *syncope*; or, lastly. by nervous exhaustion.

Asphyxia may be the consequence of inward convulsions, or of the great seizures.

In the former case, it is brought on in two very different ways. It may be immediate, as when the child dies *choked*, as if strangled, or as if his chest were violently and suddenly compressed by an iron hoop. This occurs in thymic asthma, in convulsions of the diaphragm, when the tonic contractions persist for more than a minute and a half, or two minutes, at the most, and thus completely arrest the respiratory movements, and suspend the function of an apparatus, the exercise of which is immediately necessary for the maintenance of life.

A young child, of whom I have often spoken in this lecture, died in that manner, and I shall now relate in detail the history of his case:—

He was eleven months old when he was admitted into the Necker Hospital, to which I was then attached, and placed in cot No. 11, St. Julia Ward, under my care. He was suffering from chronic diarrhœa, which improved under the influence of small doses of calomel combined with opium. In other respects the child, who was nursed by his own mother, exhibited no extraordinary symptom, when he was suddenly seized one night with eclampsia, without any premonitory symptoms. The right arm alone was convulsed, and after all slightly so. Inspiration, however, was attended with a kind of sob, somewhat similar to that of hooping-cough. These accidents, which recurred at pretty short intervals, still persisted as I was going round the ward, and returned several times in my presence, each attack lasting less than a minute, without producing any notable dyspnœa. After I had seen the other patients, I returned to the child, and had him completely stripped, and held by a nurse, so as to examine him carefully. He was then suddenly seized with tonic convulsions of the right arm, whilst his respiration quickened, and was attended with the kind of noise I mentioned just now. Within eight or ten seconds his arms and legs, and his whole trunk, became the seats of tetanic rigidity, analogous to that which obtains in the first stage of an epileptic fit. His chest-walls were fixed and motionless, his diaphragm did not move, and breathing was completely arrested. I was looking on with the greatest anxiety, impatiently waiting for clonic movements, or the least muscular twitching, when, after less than a minute of complete immobility, I saw the skin, which until then had retained its normal hue, turn livid, the face swell, and the enlarged tongue protrude out of the mouth, driving out some froth, and the urine flow copiously. I endeavoured to excite respiratory movements by squeezing and rubbing the chest, but my efforts failed, and the child died.

On making a post-mortem examination, I found slight injection of the pia-mater, as well as of the grey substance, and perhaps some slight softening of the brain, a condition which might be explained by the high temperature of the season. The most minute examination disclosed no lesion. The thymus gland was slightly larger than it usually is, but was neither indurated nor injected, and did not in the least compress the trachea. The lungs were merely engorged and full of black blood, and the bronchi contained some froth. One of the bronchial glands was slightly swollen and softened.

The second mode in which inward convulsions bring on asphyxia is perfectly different. The convulsions are complete, but the alternate contraction and relaxation of the respiratory muscles succeed one another at such short intervals, that they do not allow the chest, and consequently the lungs, to expand

sufficiently; and from spasm of the upper opening of the larynx recurring almost uninterruptedly, the air can no longer pass freely into the larynx, trachea, and bronchi. The blood is no longer regularly aërated; because, on the one hand, the respiratory apparatus no longer receives a sufficient quantity of pure air, and, on the other hand, it cannot get rid of the air which has lost its oxygen, and has therefore become useless for respiration. This function is in consequence insufficiently and incompletely performed, and asphyxia supervenes, as in cases of organic diseases of the larynx, in œdematous laryngitis, for example.

Death by the lungs may also take place in the great seizures, although less immediately than in the two preceding cases. As Dr. Duclos justly remarks, the mode in which the fatal termination occurs is somewhat analogous to what happens so frequently after tracheotomy when performed in the last stage of croup. It would seem as if all danger had been removed after an opening has been made in the trachea through which air may freely pass on to the lungs. Yet asphyxia continues, or at least we can no longer prevent the effects resulting from too prolonged disorders of hæmatosis—effects which the beautiful experiments made by Dr. Faure have so clearly shown. The patient has received a death-blow, and although the mechanical obstacle which has been the primary cause of the asphyxia is removed, we are powerless in bringing on resuscitation.

Now, children die in the same manner after convulsions which have recurred for several hours almost without interruption, and especially after that condition termed *status convulsivus*. Such repeated convulsions bring on a considerable disturbance of respiration and circulation. The face gets congested and becomes of a livid red hue; dyspnœa sets in and goes on increasing; scarcely is one paroxysm over before another comes on, followed by a third, so that respiration and circulation have not time to resume their regular course. Hence, when the attack is over and quiet has been restored, even when respiration appears to be regular, it is a deceptive calm, and the patient dies within a few hours, although there have been no fresh convulsions, no marked dyspnœa, no manifestation of grave symptoms. He dies, if I may say so, not of asphyxia, but of the sequelæ of asphyxia.

The *cerebral congestion*, which is an effect and not a cause of eclampsia, may present a certain amount of gravity, when carried to a very high degree. But although this accident has long been, and is still, regarded by some as very common and habitual, it occurs, on the contrary, in very exceptional cases.

Death by asphyxia is the usual mode of fatal termination of convulsions. In some cases, however, it must be admitted that the individuals die by *syncope*, whether this be explained by

the considerable shock to the nervous system, or by convulsions of the heart impeding its action.

Nothing is so difficult, to my mind, gentlemen, as to speak generally of *prognosis* in infantile convulsions. Prognosis in such cases is subordinate to a great many circumstances. From what I have said, you could see that inward convulsions are much more dangerous than violent convulsive seizures almost exclusively limited to the limbs. With regard to the former, there are distinctions to make between incomplete convulsions in which there are only tonic contractions lasting beyond measure, and complete convulsions made up of alternate rigidity and relaxation of the muscles. As to the great seizures, they vary in regard of intensity, duration, and more or less frequent recurrence.

In convulsions which come on at the outset, in the course, or at the close of certain complaints, it is of the highest importance to take into account the period at which they occur, as this influences prognosis considerably. I may here repeat what I have told you elsewhere. If we analyze the phenomena of which shivering consists, we find that it is, after all, a convulsion of a small degree. Whether it be partial or general, it is characterized by trembling and by involuntary movements of the parts which it affects, due to alternate contraction and relaxation of the muscles. It is not extraordinary, therefore, that these phenomena should be exaggerated in individuals whose nervous system is excitable, as it is in children, and should even pass into a true attack of eclampsia. Hence, in infancy, especially in cases of extreme nervous excitability, the slightest fever is ushered in by convulsions, whether the fever be due to a mere gastric disturbance or to some catarrhal affection, an intestinal or a pulmonary inflammation, or whether it be one of the prodromata of some continued fever.

Such *premonitory convulsions* are most frequent at the outset of eruptive fevers, of measles in particular, and still more frequently of small-pox. In fact, they are so common in such cases, that some authors, and Sydenham among others, have laid it down as an almost absolute law, that convulsions, occurring in a child who has cut his first teeth, should make one suspect the imminence of an exanthem. Sydenham, moreover, thought that such convulsions were a favourable symptom, showing that the eruptive fever would be mild.

I am far from agreeing with him on this point. Although I admit that convulsions occurring at the outset of measles and small-pox are nearly always unattended with danger, I yet believe, first, that they give no indication as to the future course of the disease, and secondly, that they may even prove (although in exceptional cases) dangerous complications, either from their

violence and frequency, or from their seat; but what exceptionally renders them serious, is uncalled-for medical interference. How often non-professional persons and even medical men, have recourse, in cases of infantile convulsions, to treatment which is always perturbing and too active. Leeches are applied behind the ears, in order to remove congestion of the brain, which is dreaded above all things, and the loss of blood, contrary to the end in view, brings the patient into a condition which is the most favourable for the production of nervous accidents. Or baths are had recourse to, cold affusions, the application of ice to the head, which, if the case be one of measles, for instance, increase the bronchial inflammation which is usually present in such cases, and change into grave complications these generally unimportant epiphenomena. Or, again, blisters are applied to the limbs, or cloths wrung out of boiling water; and the pain caused by such brutal measures excites a nervous system, which should above all be quieted.

If, on the average, convulsions occurring at the outset of diseases be generally unattended with risks, the same thing cannot be said of convulsions which come on during the acute stage of a complaint, and *à fortiori, towards its close.* They then indicate a fatal termination. Whether the case be one of pulmonary or intestinal inflammation; or of measles, whooping-cough, or small-pox, convulsions occurring in the course or towards the close of the disease, point to a danger arising from some grave complication in the patient's condition. The convulsive seizure is then preceded by brain symptoms similar to those observed in typhoid fever; it recurs for two, three, or four days, lasting sometimes from a few minutes to a few hours only, and generally ushers in death.

Such accidents are to be most dreaded in scarlatina. Even when they occur at the outset of this exanthematous fever, they have a much more serious import than when they come on at the beginning of an attack of measles or of small-pox; but when they happen in the third stage of scarlet fever, they end almost always fatally. They depend, in most cases, then, on the presence of general anasarca and concomitant albiminuria; but they manifest themselves occasionally also independently of all serous infiltration in the same manner as jactitation, delirium, vomiting, and other nervous disorders occur in the course of scarlet fever.

Prognosis, in infantile convulsions, depends on other considerations, which a practitioner should be aware of, and should take into account, in addition to the seat and the course of the convulsions, and the period at which they appear in the course of various diseases.

Clinical experience has made out the fact that convulsions

are less dangerous in proportion as they are more easily excited; and what Stoll has said of children in general, may be applied to individuals of heightened nervous excitability:—"*Convulsio et spasmus, uti frequentior in infantibus, ita minus periculosus iis plerumque est quam adultis.*" For there are individuals, indeed, who are seized with convulsions for the least thing, and in whom no unpleasant consequences follow.

Yet bear in mind that this nervous excitability may be hereditary, and that if in infancy it brings on eclampsia, it may subsequently manifest itself by producing very grave nervous affections, such as epilepsy. Recall to mind the cases which I related to you when on the subject of epilepsy, and remember especially that convulsions are accidents which expose medical men to the most unpleasant disappointments. Even those which come on under the most favourable circumstances may terminate fatally, and whenever, therefore, you are sent for to see a child seized with eclampsia, be prudently reserved.

From what I have just said, it might seem that a medical man should always interfere, and at any cost, in cases of infantile convulsions. I hold a perfectly contrary opinion. I very strongly believe that the less we do is in general the best we can do, and that our *treatment* should be expectant. If you question mothers whose children have more than once been seized with eclampsia, they will often tell you that they stopped the fit either by putting salt into the child's mouth, or by making him smell vinegar or distilled orange-flower water, or by throwing cold water in his face, or by some other method as insignificant as the above. But because medical interference is rarely called for, it must not be inferred that we must stand with folded arms in all possible cases. In convulsive seizures we should certainly be on the watch, although perturbing measures, such as bleeding, leeches, pretended revulsives to the skin, are always dangerous and almost never useful; it is essential, however, that the patient should never be lost sight of. If the progress, duration, and seat of the convulsions do not indicate danger, certain measures should be had recourse to which, without increasing the patient's risks, console his friends, sustain their hopes, and may gain for the medical man the credit of the cure. Some of these measures are, besides, unquestionably useful; and antispasmodics rank first among them, such as *ether*, either alone or combined with *musk* or belladonna, from 5 to 6 or 8 grains of musk, or from $\frac{1}{5}$th to $\frac{2}{5}$ths of a grain of belladonna.

When the convulsions keep recurring, their cause must be above all sought for; and it will be sufficient in many cases to remove the cause in order to cure the convulsions.

The timely administration of an emetic and of a purgative

enema, has been known to stop convulsions due to embarrassment of the *primæ viæ*; in other instances the fit has ceased on removing the child's clothes, when a pin stuck in badly, or too tight a bandage, was the exciting cause of the seizure.

But when the cause of the convulsions escapes us, or when it is beyond the reach of our active measures, as in eclampsia due to the pain of teething, and in certain symptomatic convulsions, there are still powerful and efficacious therapeutic measures which may be used against the convulsions when they are prolonged. Such are *compression of the carotids* and *chloroform inhalations*.

You are aware how prudently chloroform should be used, how you should keep your finger on the patient's pulse, counting the number and feeling the strength of the pulsations; and by taking these indispensable precautions you may be able to push on the inhalation very far. In the beginning of the year 1860, I was sent for to see a child, five years of age, the son of one of my best friends, who had on the previous day had a very slight attack of convulsions. He was afflicted with disease of the brain, which had arrested his mental development. I was sent for because he had been again seized with convulsions which were this time awfully violent. When I saw him his face was congested to such a degree that he looked as if in the last stage of asphyxia. I made him inhale some chloroform poured on a handkerchief, which I held some distance from his nostrils, for a few minutes at a time, taking the precaution of constantly feeling his pulse. For six whole hours, from six to twelve o'clock, I thus administered chloroform almost without interruption, and I could not say how much I used. Thanks to this mode of treatment, the child, who was at the point of death, recovered, and is at present as well as he was formerly. I have raised, and still raise, my voice against the application of revulsives to the skin, and of blisters in particular, as they have seemed to me to do in general more harm than good. There are cases, however, in which these measures become necessary, and may be really useful, namely, cases of *inward convulsions* which involve the diaphragm and the heart itself, are of the tonic kind, and are so prolonged as to bring on asphyxia or syncope. In such instances a violent and rapid revulsion to the skin of the chest, such as can be produced by ammonia, may do good, by exciting irritation which rouses into action those muscles the play of which is indispensable for the acts of respiration and circulation.

LECTURE XII.

ECLAMPSIA OF PREGNANT AND PARTURIENT WOMEN.

Gentlemen,—The details into which I entered, in our last conference, allow me to be brief in what I have to tell you to-day of *puerperal eclampsia, à propos* of a patient who lay in bed No. 28 in St. Bernard Ward. Were I to give you a detailed description of this affection, I should have to repeat, in a great part, what I said to you about infantile convulsions. This latter description was itself singularly like that of epilepsy; for, as I have more than once told you, these affections present the greatest analogies to one another, if we merely look at their outward manifestations.

Recall to mind what happened in the case of the young woman who was in St. Bernard Ward; and those among you who witnessed her violent convulsive seizures, could see how they resembled epileptic fits.

These convulsions occurred under the following circumstances: on the day previous to her admission, the patient had been delivered, at three o'clock in the morning, of her first child. She had complained of nothing peculiar during her pregnancy. The midwife who was with her, gave her a full dose of ergot of rye after delivery, probably with the idea of stopping an abundant loss of blood. Convulsions came on two hours afterwards, and she was brought to the hospital in the course of the day.

My clinical assistant, M. Moynier, on seeing her in the evening, decided on bleeding her at the elbow, to the amount of about 27 ounces. Still, the convulsions recurred with extreme violence from 6 to 12 o'clock P.M. They had ceased when I saw the patient on the following morning. The lividity of the face, which had on the previous day been carried to a very high degree, had almost completely disappeared. The tongue bore traces of having been bitten in several places. I prescribed the following mixture:—

℞: Moschi	gr. x.
Ext. valerianæ	āā.
Aquæ minthæ	ʒ ij.
[1] Syrupi ætheris	℥ fs. āā.
„ floris aurantii	

[1] The syrup of ether of the French codex contains half a drachm of ether in each ounce of syrup.—Ed.]

About 11 A.M., next day, she had another attack, as violent as the previous ones, and followed, like them, by profound stupor and complete loss of consciousness.

The puerperal convulsions had in this case occurred at a period when they are not generally common, namely, after delivery. For, indeed, obstetric teachers tell us that eclampsia is rare before the sixth month of pregnancy, is less rare after delivery than during pregnancy, while it is most frequent during labour.

In the present case, I could not make out the *exciting cause* of the seizures, and the only etiological condition to which I could ascribe them was that the patient was a primipara. The *influence of a first pregnancy* on the production of eclampsia (as a predisposing cause) is a fact admitted by most accoucheurs. According to Cazeaux, whose work is in everybody's hands, seven out of eight cases of eclampsia occur in primiparæ. But although the influence of a first birth is so considerable, it must not be inferred that a woman, who has passed through a first pregnancy and been delivered safely, is for ever secure against puerperal convulsions, nor does the occurrence of convulsions in a previous pregnancy necessarily imply their recurrence in succeeding pregnancies.

If you remember what I told you of epilepsy and of infantile convulsions, it is unquestionable that the *nervous excitability* which, in some women, manifested itself during infancy by convulsive seizures, and later by hysterical symptoms or more less curious nervous disorders, is a predisposing cause which should engage the attention of the physician.

I shall not review all the exciting causes enumerated in text-books, but there is one to which I am anxious to call your attention, although it was absent in the case of my patient, I mean, *albuminuria*.

I need not discuss here whether the albuminuria which occurs in pregnancy be at the outset caused solely by compression of the kidneys, of the iliac veins, or the trunk of the vena cava inferior by the uterus; whether it depends, as Braun, of Vienna, believes, on this compression, and the resulting stagnation of the venous blood, and on the peculiar modifications which the blood undergoes during pregnancy; or whether it is due to the nervous disturbance which so often accompanies pregnancy.

It has been sufficiently proved by clinical observation that albuminuria occurs pretty frequently during pregnancy, especially in primiparæ, and in women who have a malformed pelvis, whose uterus is too high up or is of considerable size, either from the presence of a very large fœtus, or of several fœtuses, or of an excessive quantity of liquor amnii.

It is sufficiently proved also that this albuminuria has pretty

frequently an unfavourable influence on the course of pregnancy, and on delivery and its consequences, and lastly, although it has been denied by some, that there is a relation, a coincidence at the very least, between albuminuria and puerperal convulsions.

It should be added, it is true, that the coincidence is far from being constant.

Albuminuria stands to eclampsia in the same relation as it does to anasarca. Although anasarca and albuminuria often coexist (and there is then an evident relation between them), the former may be present without the least trace of albumen being detectible in the urine, while, *per contra*, partial or general dropsy may be completely absent, and yet there be abundant albuminuria. In the same manner, although convulsions recur very frequently in women who pass albuminous urine (Mr. Imbert-Gourbeyre has met with it 94 times out of 159)[1], and although, consequently, the presence of albuminuria during pregnancy must make one dread the occurrence of eclampsia at a more or less distant period, it must not be forgotten that, in a great many cases, convulsions never occurred, although the urine had for a long time been albuminous.

Lastly, the case of the young woman which I have related, and other instances which have come under my observation, formally disprove the law which has been laid down by some, that in all cases of eclampsia occurring in women, the urine was invariably found to contain albumen. The urine of that young woman was examined on repeated occasions, and neither heat, nor nitric acid, ever gave rise to the least albuminous cloudiness.

Most commonly, if not always, puerperal convulsions are general, as they were in the case which is still in the hospital. In some rare instances, however, they are *partial*, and the following case, which came under my care at the Necker Hospital, seems to me to present some analogies to this partial form of eclampsia.

A woman, 21 years of age, who had six months previously been delivered, at her full term, of the baby whom she was then nursing, was admitted into St. Anne Ward, and placed in bed No. 24, on January 16th, 1846. Her previous health had been good, but two months before delivery, she had been seized with convulsions, which had come on suddenly during the day, without any appreciable cause, and which, after affecting the whole left side of the body, left behind them incomplete hemiplegia, that lasted an hour. The patient was not unconscious during the attack.

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[1] De l'albuminurie puerpérale et de ses rapports avec l'éclampsie (Mémoires de l'Académie royale de Médecine. Paris, 1856, t. xx.)

She was safely delivered, but two months subsequently, and this time during the night, she had another attack, which recurred three weeks later, consisted of several fits, and lasted from half an hour to an hour. The seizures returned after this every week at first, and then every day. From December 28th or 30th, 1845, to January 16th, 1846, when I saw her, the attacks had returned nearly without intermission, and from that time, also, the left arm and leg had been paralyzed. She complained of having, in the affected limbs, a sensation unattended with pain, but which she compared to "something running over her leg;" the convulsions then began, first in the foot, then gradually extending up to the trunk, involving the arm and even the muscles of the face; at other times, they spread from above downwards, and at other times, again, they were limited to the face.

They consisted at first in tetanic rigidity and distortion of the affected limbs, almost immediately followed by convulsive jerks, and the paroxysm terminated in relaxation. Meanwhile, her health was good; she had a good appetite, and I could not find any other symptom of local disease either in the brain, or in the thoracic or abdominal organs.

Perhaps you will think, gentlemen, that the only connexion which these seizures had with eclampsia was their having come on for the first time during pregnancy, and that they did not resemble puerperal convulsions, their form reminding one rather of partial epilepsy preceded by an *aura*. I will observe to you, however, that although they were epileptiform, these attacks differed essentially from epileptic fits in their mode of invasion and their course.

The patient remained in the hospital until the following March. She was given strychnine at first, but narcotics were soon substituted for it, chiefly belladonna, which was given at once in 3-grain doses. The convulsive seizures gradually diminished in violence and frequency, and ceased completely by the 24th of February. The paralysis lasted longer; from the beginning of March, there was only a little numbness in the affected parts, and when she was discharged on March 20th, she had seemed to be perfectly cured for several days previously.

To those who might regard this case as a kind of chorea, I will answer, that St. Vitus's dance presents neither this form nor this course; that the same may be said of chorea, or, if you prefer, of hysterical trembling; lastly, that if the case cannot be absolutely regarded as an instance of *partial* eclampsia, it cannot be referred to a well-defined nosological species, and can, therefore, be mentioned in connexion with convulsions occurring in pregnant women.

But I must return to the case of the woman in St. Bernard Ward. After her attacks of eclampsia, the last of which occurred on September 11th, she remained for 48 hours in a state of profound coma; on the 13th, during the night, she became delirious and was so restless, that she had to be confined with a strait-jacket.

From the 16th to the 20th she was well, and seemed to have calmed down, there only remaining hebetude which had persisted, when on the 21st, during my visit, she had an attack of acute *mania*. She began calling out, on a sudden, "My daughter! my daughter!" with eyes bright with excitement, and asking for her child, who had been taken away from her. She seemed to be unconscious of what she was saying or doing, and still had the look of hebetude which had never left her from the beginning. On my ward being cleared out in order to be cleaned, the patient was removed into another ward, under another physician's care, and soon recovered.

Mania is a pretty common result of eclampsia, and there are instances on record in which the unfortunate women have continued in that state of maniacal delirium, and sometimes of more or less complete dementia. Generally, after an attack, the intellectual faculties are disordered for a longer or shorter period; memory is particularly impaired, and sometimes lost completely, and the patients have no recollection, for several days, not only of the seizure which they have just had, but also of the circumstances which preceded it. The loss of memory is sometimes partial only, and relates to certain subjects, such as forgetting the names of certain persons, even of those whom they see most frequently and who are dearest to them.

Paralysis is one of the most frequent of the unpleasant sequelæ of eclampsia, and it may be due to an organic lesion of the brain, such as hæmorrhage into the meninges or the substance of the brain. The same thing happens here as in epilepsy. Note that, in both cases, the cerebral congestion, which is sometimes so intense as to result in hæmorrhage, is no more the cause of the puerperal convulsions than it is that of the epileptic fit; it is an effect, and nothing more.

I therefore do not include in the *treatment* of eclampsia, *general* or *local bleeding*, intended to do away with this pretended cause of puerperal convulsions, no more than I advise them in epilepsy or in the eclampsia of children.

Antispasmodics are, on the contrary, formally indicated in such cases, and *chloroform* inhalations rank first among them. There are already a pretty large number of cases in which chloroform has unquestionably done good. By its cautious use several times in succession, violent attacks have been known to be completely arrested, and convalescence to begin imme-

diately. I shall mention in connexion with this point cases published by M. Gros in the *Bulletin général de Thérapeutique*, for January, 1849, which you will read with profit; and others, by M. Richet, in another periodical; while Dr. Campbell only recently communicated to me, among other cases which have come under his personal observation, the marvellous results which he had obtained by this method of treatment in the instance of a child, the daughter of a very high personage in the state. I will add, that many eminent accoucheurs (among whom I shall mention M. Blot by name), who had long opposed the use of chloroform in the treatment of eclampsia occurring during labour, now acknowledge, and strongly advocate, the use of this heroic remedy.

I will, in conclusion, remind you that when eclampsia comes on in the eighth or ninth month of pregnancy, and has resisted all treatment, the induction of premature labour is adopted by most accoucheurs, a measure recommended by Stoltz, and approved of by men of the highest authority, among whom I need only mention Professor Velpeau and Dr. Cazeaux.[1] When eclampsia occurs during labour, delivery should be hastened, if the attacks be violent, in order to save the mother and the child from the danger which they incur. Still, gentlemen, although the convulsions cease soon after delivery in the majority of instances, they continue to recur with renewed intensity in some cases, and quickly terminate fatally.

[1] [See a paper on the "Induction of Premature Labour in Cases of Pregnancy complicated with Albuminous Urine, Dropsy, and Amaurosis," by Robert Lee, M.D. in "Medico-Chirurgical Transactions." Vol. xlvi. p. 169.—Ed.]

LECTURE XIII.

ON TETANY.

Causes: the most frequent are nursing and the puerperal state; influence of antecedent diarrhœa; effect of cold.—Description of the disease: three arbitrary forms.—Mild form: local manifestations are alone present, and the symptoms are very slight.—Intermediate form: the contractions become general, and spread from the extremities to the muscles of the trunk and face, while general symptoms are superadded to them.—Grave form: violence of the convulsions.—A fatal case.—Prognosis generally not grave. Pathological anatomy very little known. Nature of the disease.—Differential diagnosis.—Treatment.

Gentlemen,—I shall devote this conference to the clinical study of a strange complaint of which I have had the opportunity of showing you instances in my wards; and which has been in turn called *intermittent tetanus*, *idiopathic contraction* and *paralysis*, *idiopathic muscular spasm*, *contraction of the extremities*, and, which I myself called at one time, *rheumatic contraction of nurses*, but prefer calling now, for reasons which I shall presently tell you, *intermittent rheumatic contractions*, and still better, *tetany*.

This complaint is in general of no gravity, although it sometimes frightens the patients who suffer from it, and misleads medical men who do not recognize it; and it is developed under circumstances which are so frequently met with, and under the influence of such common causes, that it must have always existed. Yet, whether it was unobserved, or rather whether the phenomena which characterize it were confounded with other forms of convulsive disorders, there is no description of it to be found in old writers; and we scarcely find, scattered through their writings, a few cases presenting some analogy to those which we observe nowadays. The history of tetany dates, therefore, from our own time; and, indeed, it is only for the last thirty years, and especially for the last few years, that attention has been particularly directed to it.

In 1831, a memoir was published by Dance, in the *Archives générales de Médecine*, with this title: "Observations sur une espèce de tétanos intermittent" ("Observations on a form of intermittent tetanus"). It was the first essay on the subject, and was soon followed by memoirs written by Tonnelé,[1]

[1] Mémoire sur une nouvelle maladie convulsive des enfants. (Gazette Médicale, t. iii. no. 1. 1832.)

Constant,[1] Murdoch,[2] and de la Berge.[3] Since then tetany had henceforth a place assigned to it in textbooks; or at least Rilliet and Barthez, in their special treatises on Diseases of Children, and Monneret and de la Berge, in the "Compendium of Practical Medicine," devoted important articles to it. In 1843 a memoir was published in the "Journal de Médecine," by Messrs. Teissier and Hermel, on Idiopathic Contraction and Paralysis in Adults ("De la contracture et de la paralysie idiopathiques chez les adultes"); and in the following year, Dr. Imbert-Gourbeyre, now Professor in the Preparatory Medical School at Clermont-Ferrand, chose for the subject of his inaugural address, *De la Contracture des Extrémités* (Contraction of the Extremities). Numerous cases had been observed, several of which had been published in various medical journals; and I had myself collected a pretty imposing number of them at the Necker Hospital, when, in 1846, my friend Dr. Delpech, then my clinical assistant, now my colleague and Assistant Professor in the Faculty of Medicine, wrote a thesis on *Idiopathic Muscular Spasms*, in which he summed up with talent all that had been done before, and added other cases to those already known. Six years later, Dr. Lucien Corvisart took up the same subject, and proposed the name of *Tetany* as a substitute for that of *Contraction of the Extremities.*

In 1855 a communication from Aran to the Medical Society of the Paris Hospitals gave rise to an interesting discussion on the disease in question. Lastly, still more recently, Dr. Rabaud, house-physician to the St. Antoine Hospital, published his *Recherches sur l'histoire et les causes des contractures des extrémités* ("Researches into the History and the Causes of Contractions of the Extremities.") The author of this monograph, however (which is lengthy and conscientiously written), committed the geat fault of confounding together all kinds of contraction.

The complaint, of which I am going to speak, constitutes a very distinct species. The conditions under which it is developed, the causes which seem to bring it on, the form which it assumes, and its course, are all of them well-defined characters.

As the first cases which I saw at the Necker Hospital occurred in women who had been recently confined and who were nursing, I at first thought that the disease was special to nurses, and I therefore called it *Rheumatic Contraction occurring in Nurses*; but it was not long before I found out, what others had besides said

[1] "Observations et réflexions sur les contractures essentielles." (*Gazette Médicale*, p. 80, 1832; et *Bulletin de Thérapeutique*, 1835.)

[2] "Considérations sur les rétractions musculaires et spasmodiques." (*Journal Hebdomadaire*, t. viii. 1832, p. 417.)

[3] "Note sur certaines rétractions musculaires de courte durée," etc. (*Journal Hebdomadaire des Progrès, etc.*, t. iv. 1835.)

before me—namely, that nursing was not the only *favourable condition for its development.*

It must be acknowledged, however, that *nursing* is, perhaps, the most frequent and active cause of intermittent contractions. I shall not attempt to explain why and how this is so; but clinical experience establishes the fact, and, judging from what happens under our own eyes, its influence is unquestionable, since in that portion of St. Bernard Ward which is reserved for wet-nurses, and contains twelve beds only, we have always seen a greater number of cases of this complaint than in all the other wards.

Menstruation, the *puerperal state*, and *pregnancy* especially, have been ascribed as causes; and one may admit that there is a connection between the phenomena of tetany and other nervous disorders which are so frequently met with in those intermediate conditions between health and disease; yet contractions occur not only in women apart from such conditions, but even in individuals of the other *sex.*

They most commonly occur in young people, and in the majority of my cases, the patients' age ranged from 17 to 30. A woman, however, who was in bed No. 20 in St. Bernard Ward, was 46 years old. She had been delivered two months previously, and the symptoms in her case were rather marked. Instances of individuals thus affected are on record, who were 52 and even 60 years old. It is not uncommon to see the complaint in children, and even in infants from 1 to 2 years old; and you may recollect seeing a very remarkable case, that of a little girl 21 months old.

She was the eighth child of a woman aged 30. Soon after birth she had violent attacks of eclampsia, and was still subject to partial convulsions, consisting in spasmodic trembling of the upper eyelid and the globe of the eye; sometimes in spasm of the glottis, which came on under the influence of emotion or of a feeling of annoyance, and was characterized by a prolonged and whistling inspiration. Contractions of the extremities (phenomena of the same nature as the preceding) were very marked; the thumb was forcibly adducted, and flexed into the palm of the hand underneath the fingers, which were pressed against one another. There was œdema of the feet and of the upper limbs of the same degree. The child was of weakly constitution, and was suffering from membranous ulcerative stomatitis; the exudations, which were of a greyish-white colour, extended over her tongue; and she had also had, for the last nine months, a cough which for some time past had assumed a convulsive character.

The process of *teething*, which so manifestly predisposes, either directly or indirectly, to convulsions, has been regarded as

exerting an influence also on the development of tetany. But it may be conceived how difficult it is to appreciate such a cause as this, particularly as it is nearly always complicated with various pathological conditions, on which tetany would seem rather to depend.

Of these pathological conditions, *diarrhœa*, especially when abundant and chronic, is the one which exerts the most striking influence. This *exciting* cause had at first completely escaped my attention. My friend Dr. Lasègue was the first who clearly pointed it out, and since then it has been mentioned by others, especially by Aran. Its influence is now admitted by all practitioners in the majority of cases, and you have been able yourselves, by questioning the patients, to ascertain that it is almost constantly present.

Yet in a young man who was in St. Agnes Ward, contractions coexisted with obstinate constipation, and disappeared, on the contrary, when the bowels were freely acted on by purgatives.

This patient was stout and well-developed, a saddler by trade, and 21 years of age; he remained in hospital about five weeks. His complaint dated four years back. His health had been good until then, and he was seized suddenly, and for the first time, while travelling by rail. Although it was winter-time, he affirmed that he had not caught cold. He noticed suddenly that his fingers kept bent, and that he could not extend and use them. This lasted for two or three hours, and recurred every day for three months, his general health being, after all, unaffected. He was treated by bleedings, but immediately after each bleeding, the contractions were not only more violent, but became general also, involving the muscles of the limbs, trunk, and face, to such a degree that his respiration was impeded and his speech embarrassed. In proportion also as the bleedings were repeated, the fits became more violent, so much so, that they were never so bad as after the fourth time of bleeding. Yet, by cupping him in twelve different places along the vertebral column, a perfectly different result was obtained, for the contractions disappeared for the period of ten months. After that time they returned, and then recurred every year, coming on every day for two months, and always at the end of winter. During the summer previous to his admission into the hospital, he had two or three attacks, transient only, and so slight that he was not obliged to give up his occupation. His general health was good all the while; he had a good appetite; but I wish to draw your attention to the fact, that his bowels, instead of being regular as formerly, were *obstinately costive*. By taking nearly two ounces of epsom-salts, once a week, he managed to unload his bowels, and thus to remove his con-

tractions for a time; but the costiveness returned as badly as ever, and his bowels did not act for four or five days.

This is too exceptional a case to invalidate in the least the general law which may be laid down regarding the influence of diarrhœa on the production of intermittent contractions.

These may come on also after a severe illness, and in the cholera epidemic of 1854, I met with many cases in individuals who had suffered from cholera. They may occur after grave fevers, as typhoid fever in particular, as M. Demarquay (quoted by M. Imbert-Gourbeyre) and Dr. Delpech have recorded instances.

Perhaps some may ascribe the disease in such cases to the intestinal flux, which is such a predominating symptom in cholera and typhoid fever; but I will remind them in answer, that contractions occur equally, although less frequently, in individuals who are convalescent from diseases in which diarrhœa is not a usual symptom, or in which it is only a temporary epiphenomenon of no great value; and that the muscular spasms must therefore be more justly regarded as accidents of the same nature as the nervous phenomena, the paralysis, &c., which prolonged illness (grave fevers especially) leave behind them, and which result either from a direct action of the morbid cause on the nervous centres, or from the nervous erethism which coexists with general enfeeblement of the system.

Besides the above predisposing causes, there are some *exciting* ones which I shall point out to you. The influence of *emotion*, mentioned by authors, is very doubtful, in my opinion, at least as far as the first attack is concerned; for I admit that when a person is already subject to contractions, emotion may bring back an attack.

A woman, 21 years old, who was in bed No. 11 in St. Bernard Ward, and who was seized with contractions in the fifth month of her pregnancy, had more violent attacks when under the influence of emotion.

If this kind of causes should not be accepted without reserve, the same does not apply to *cold*, the influence of which has been pointed out by all observers, and which unquestionably acts not only as an exciting cause, but is sufficient by itself to bring on the disease.

I will relate a few instances in point. A patient in St. Agnes Ward ascribed his complaint to his catching cold on going out one December day too thinly clad for the season, and he had felt the cold all the more keenly, that he was in the habit of working in a very heated room.

Another, at No. 23 in the same ward, had spent the night out-of-doors in a stake of drunkenness, and had been found the next morning in the state in which he was sent to the hospital from the police-station.

A woman, to whose case I shall revert by-and-by, was seized with contractions after having, during winter nights, fetched water from the hospital yard. The cold had great influence on her, because she had been recently confined prematurely, was weakened by poverty, and by an obstinate diarrhœa which had scarcely left her.

Lastly, when I describe to you the phenomena which characterize contractions, I shall show you that compression of the affected limb brings them on very rapidly and without fail.

It is no easy task to draw a sketch of this complaint, and the best description cannot give you an idea of what you could not forget when you had once seen it. I will still endeavour to give you as accurate an idea of it as possible, and in order to enable you to see its principal features better, I shall speak of three distinct forms of the disease, although these divisions are in reality quite arbitrary.

In the first form, which I will call the *mild form*, there are only *local manifestations*, and they are as follows :—

The person has a sensation of tingling in the hands and feet, and then feels some hesitation, some impediment in the movements of his fingers and toes, which are not as free as usual. Tonic convulsions then set in, the affected limbs become stiff, and the will cannot completely overcome this stiffness, although it still struggles with it, and the patient can still use, within certain limits, the contracted muscles, move and even extend his fingers. The involuntary contraction increases, becomes painful, and is exactly like a cramp to which the patient compares it besides.

In the upper limbs, the thumb is forcibly and violently adducted; the fingers are pressed closely together, and semi-flexed over the thumb in consequence of the flexion of the metacarpo-phalangeal articulation; and the palm of the hand being made hollow by the approximation of its outer and inner margins, the hand assumes a conical shape, or better the shape which the accoucheur gives to it when introducing it into the vagina. This aspect of the hand, which you will most frequently meet with, is so peculiar that it is oftentimes sufficient by itself to characterize this kind of contraction. In some cases the index-finger is more powerfully flexed than the other fingers, and is partially bent under them; in other cases, the flexion is more general and complete. The thumb is turned into the palm and hidden by the fingers, which are themselves bent, and with such force that the nails leave an imprint on the skin; and they are so squeezed together, that in a case recorded by Dr. Hérard, sloughs actually resulted from the prolonged pressure. The thumb alone may be affected, while the fingers are scarcely contracted; but such cases are rare, and it more commonly

happens that the contractions spread to other parts, the wrist becoming flexed, and the hand turning forcibly inwards, the patient having lost the power of straightening it.

In the lower limbs, the toes are bent down towards the sole, and press against one another, while the big toe turns in under them, and the sole becomes hollowed out in the same manner as the hand. The dorsum of the foot is strongly arched, and the heel pulled up by the contracted muscles at the back of the leg, while the leg itself and the thigh are in a state of extension. The contractions may affect the upper and lower limbs simultaneously, or alternately; or they may be confined to one of them. In exceptional cases the lower limbs are alone involved, while most commonly the hands are the parts that are affected. The convulsed muscles resist the efforts that are made to alter the position which they make the parts assume; and if their resistance be overcome, the fingers bend again as soon as they are let free, or, in exceptional cases, they keep the last position in which they are placed, although remaining contracted all the while. To the touch the muscles feel more or less hard, like tense and rigid ropes; but I have never, for my part, felt the fibrillary contractions which have been spoken of by some. The efforts made to overcome their resistance give the patient pain, although some relief is thus procured in certain cases.

These tonic convulsions last uninterruptedly for five, ten, or fifteen minutes, and sometimes even one, two, and three hours in succession; the sensation of formication then returns, and announces the termination of the attack in the same manner as it ushered it in. The affected parts become moveable again, until, after a variable interval of rest, fresh paroxysms recur, the series of which constitutes the attack, which may be prolonged for several days, and even for one, two, and three months. So long as the attack is not over, the paroxysms may be reproduced at will, even though the patient has been free from them for 24, 36, 48, 72 hours, and more.

This is effected by simply *compressing the affected parts, either in the direction of their principal nerve-trunks, or over their bloodvessels, so as to impede the venous or arterial circulation.*

I discovered this influence of pressure by chance. I was present when a woman suffering from contractions was being bled from the arm at the Necker Hospital, and I saw a paroxysm return in the hand on the same side when the bandage was applied round the arm. I at first thought that it was brought on by the venous congestion caused by the pressure on the vein; but on trying to account for the phenomenon, I found in other patients that by compressing the arteries, the same results were produced. I have often since repeated the experiment, and as the contractions cease as soon as the pressure is

removed, and the patient is therefore not much troubled, I have often made it in your presence. You saw then, that not only when the arterial or venous circulation was interrupted, but also when the median nerve was compressed in the arm, or the brachial plexus above the clavicle, the contractions came on, immediately preceded by the sensation of formication, which is its first symptom. When the femoral artery is compressed, as when a ligature is applied round the thigh, or when the limb is firmly squeezed between both hands, and the sciatic nerve thus compressed, spasms of the muscles of the lower extremities are brought on, although with less facility. This phenomenon, which is already interesting by itself, is not besides without its practical utility; for it furnishes us with a means of diagnosis, as in no other convulsive disease are such effects produced by similar means.

It is an extraordinary circumstance that *cold* (which has so manifest an influence on the development of this complaint) *should sometimes stop the contractions when applied to the affected parts.* Thus it happens that, in a great many cases, patients suffering from contractions of the lower limbs have only to stand with naked feet on a stone or tiled floor in order to stop the convulsion almost instantly, and to regain the free use of their limbs. I have, in many instances, arrested paroxysms of contraction in the upper extremities, by making the patient dip his arms and hands into a basin of cold water. The arrest is only temporary, it is true, and the contraction returns when the part is no longer immersed.

Intermittent contractions are generally preceded and accompanied by loss of muscular power. Movements of extension are not the only ones abolished by the convulsive contraction of the muscles; those of flexion are equally so. The fingers, for instance, when half flexed, no longer obey the will, and the patient cannot close them further. This rigidity is sometimes, in grave cases, carried to a very high degree; but even when it is slight, as in mild cases, and is added to the convulsive stiffness of the hands, it renders the patients clumsy, and prevents them from freely using their hands; if they are nursing at the time, they cannot attend to the child in the usual way, dress or even hold him in their arms.

There is *anæsthesia* besides, and the sensations of formication, tingling, and numbness are referable to it. The sense of touch is more or less impaired, so that the patients lose the faculty of appreciating the size and hardness of the objects which they take hold of, and which feel to them as if wrapped in some thick material. When they walk, they have the same sensation as if they were walking on a carpet. Now these alterations of cutaneous sensibility, the integrity of which is so

necessary for the regularity of muscular functions, concur in impeding movements.

I have told you already that these contractions are generally attended with pain, which is seated in the affected muscles, extends along the course of the limbs in the direction of the principal nerves, and radiate sometimes to the trunk. This pain, the presence of which by no means excludes anæsthesia, is often very moderate in the mild form of the complaint; and as, on the other hand, the convulsive phenomena are often very transitory, the result is that the patient does not complain, and, in some instances, chance alone makes us discover their ailment. This was the case, among others, with a woman who occupied bed 20 in St. Bernard Ward. She was admitted into the hospital for a diarrhœa of somewhat old date; and if I had not, on going round the ward, witnessed myself an attack of contraction in her hands, she would never have thought of complaining of an ailment of which she took no notice whatever.

The case is perfectly different in the other two forms.

In that of medium intensity, the violence of the pain and of the spasm is more marked; and the local manifestations are, besides, complicated with *general symptoms*, such as febrile excitement characterized by an acceleration of the pulse, malaise, cephalalgia, and loss of appetite. The fever, however, never runs very high, and is never accompanied by a marked heat of skin. Transient congestion happens in different parts of the body—in the limbs, the face, the eyes, and ears; sometimes they are accompanied by dizziness, obnubilatio, and tinnitus aurium.

This congestive process sometimes causes swelling and œdematous puffiness of the limbs, which have been mentioned in several cases, principally in children. As to the contractions themselves, they are not only stronger than in the mild form, and return more frequently, but they are general also, instead of being confined to the extremities, and involve the muscles of the trunk and face, and sometimes also those of organic life.

The spasms do not, as a rule, involve simultaneously the muscles of the trunk and extremities. The upper extremities are generally the first to be affected, and while the antecedent numbness and formication descended from the arm to the hand, the convulsions, following a reverse course, begin in the fingers, and successively extend to the wrist and elbow. The lower limbs are rarely seized before the upper. From the extremities, the contractions spread to other portions of the body, and the short time during which they last in a given part, in fact, the mobility of the complaint, is a character of which I shall make use when I come to speak of its nature.

The abdominal muscles may be affected, and in a case published by Dr. Hérard, the recti muscles stood out like two very tense cords. Instances have also been recorded in which the spasm has extended to the bladder, and caused retention of urine. The pectorales majores and sterno-cleido-mastoidei have been seen to contract violently, and it is not uncommon to see tonic convulsions of the face. The patient's aspect is then very peculiar, according to the set of muscles which are affected; when the muscles of the eyeball are convulsed, strabismus results, either external or internal—that is, divergent or convergent. At other times the jaws are firmly clenched, and the embarrassment of speech may also be due to the tongue being involved. Deglutition is impeded when the pharynx is affected, and when the larynx is involved, the series of symptoms of thymic asthma are produced, as in the case of the little girl which I related at the beginning of the lecture. The laryngeal spasm, and the contraction of the muscles of the abdomen and chest, bring on more or less marked dyspnœa, which becomes extreme when the diaphragm is involved.

The third and *grave* form of the complaint is characterized by the prolonged duration of the contractions, their recurrence after short intervals, and their greater intensity. In the month of December 1856, my colleague and friend, Dr. Lasègue, was consulted about a patient who was believed to be epileptic, at the Prefecture of Police, where he goes every day, as he is physician to the department for the insane there. The patient was that young man, 18 years of age, whom you afterwards saw at No. 13 in St. Agnes Ward, and to whose case I have already alluded. He had been found in the morning lying down in the streets, where he had spent the night in a state of drunkenness. All his muscles seemed to be in a state of violent contraction, and he was as stiff as a poker: he was perfectly conscious, however; and although his speech was considerably embarrassed, through his inability to open his clenched jaws, he gave distinct answers to the questions that were put to him, and complained of great pain. The continuance of this general tonic convulsion, and the perfect preservation of the intellect, excluded at once all idea of apoplexy; while the character of the symptoms, and especially of those of the upper limbs (the hands being in the peculiar attitude which I have endeavoured to describe to you), allowed Dr. Lasègue to diagnose the case immediately, so that he had him sent to the Hôtel-Dieu. The interval between the intermittent paroxysms was very short. All his muscles, those of the trunk and of the cervical region as well as those of the limbs, seemed to be simultaneously affected, and, unable to move at all, the patient fell down in a condition of tetanic rigidity. The contractions were very

painful, and after a short time the breathing became embarrassed, from the tonic convulsion of the muscles of the chest, abdomen, and diaphragm, the larynx itself not being spared. The face became red, and the lips livid; the veins swelled, and during this fit of awful dyspnœa, attended with pulmonary engorgement, as in epilepsy, or better as in tetanus, suffocation was to be dreaded. Fortunately, this state of things lasted a very short time only.

You have more than once witnessed these attacks, when going round with me. They came on suddenly, ushered in by a sensation of formication, and lasted several minutes, sometimes for a quarter of an hour, or even half an hour. The mind of the unfortunate patient was perfectly clear, and he could speak, although the contracted state of the muscles of the jaws embarrassed his speech considerably; even in spite of his pain, he was somewhat cheerful.

When the attack was over, he got out of bed and went on with his occupation, rendering slight services to his companions, and acting as sick attendant. In the intervals between the attacks his general health seemed by no means disturbed. The attacks, however, left behind them lumbar pain and a feeling of contusion (in the joints chiefly), and a state of weakness and prostration which lasted for some time. On several occasions I noted some fever.

The seizures became by degrees more rare, and after remaining a month and a half in hospital he wished to go home. But six weeks afterwards he was seized in the same manner again, and was readmitted into the Hôtel-Dieu, under the care of my esteemed colleague, Professor Rostan, in whose ward he died of pulmonary consumption. The tubercular disease remained latent to the last, and was not revealed by stethoscopic signs; this peculiarity was dwelt upon in the notes given me of this patient's case; the general debility, cough, and habitual dyspnœa alone caused it to be suspected. On a post-mortem examination, the lungs were found to be infiltrated with tubercles, and the spinal cord to be slightly softened at its upper part.

I shall return to this case presently, when I tell you my views of the nature of tetany, and speak of the relations between it and the anatomical lesions which have been found. I will then tell you that the convulsive seizures in this case were by no means due to the tubercles, of which no material manifestation was found in the nervous centres, and that the softening of the cord should be regarded not as the cause but an effect of the disease. I shall enter into explanations on this point, which I have besides dwelt upon in our conferences on Apoplexy and on Convulsions.

I do not admit the supposition that the contractions were in this case due to the tubercular diathesis, and this cannot be put forward in the following case observed, in M. Cullerier's wards, by Dr. Blondeau, whilst a resident assistant at Lourcine Hospital.

Elizabeth B——, aged 28, was admitted January 20, 1848, into St. Mary's Ward, No. 32. She was in the eighth month of pregnancy, and was suffering from syphilis, with numerous ulcerated mucous tubercles in the external organs of generation. She had, besides, a very copious and obstinate diarrhœa. She was in a state of considerable weakness and marasmus, and on February 13 she was delivered of a stillborn child. Two slight contractions of the uterus, which were scarcely perceived, were sufficient to expel the fœtus. The diarrhœa ceased at last, on the administration of nitrate of silver injections. It was completely arrested five days after delivery; the appetite became good again, all the digestive functions regular, and a marked improvement of the general condition of the patient was observed from day to day. She had even regained her strength and a certain amount of flesh, when she was seized, on February 27, with symptoms which terminated fatally.

She complained in the morning of some swelling of the feet, and expressed a fear that she might be again paralysed, as she had been on a former occasion. She added, however, that she felt well, and indeed, apart from this slight swelling of the lower limbs, nothing was found which called attention. On the following night she had a violent pain in the head, and the next morning she was seized with tetany.

Her hands and feet were violently convulsed, and her fingers and toes semiflexed, in the attitude which I need not again describe. The muscles were so contracted that all efforts to overcome their resistance proved useless. The muscles of the face were involved, the jaws were convulsively clenched, and speech was embarrassed. The patient, however, still answered the questions that were put to her, and her intellect was perfect; as the muscles of the neck and chest shared in this general convulsion, respiration was impeded, and the face red and congested.

It was then ascertained that the patient since her delivery, and even when her diarrhœa had scarcely stopped, had on several occasions got out of bed during the night, and fetched water from the fountain in the hospital yard. On the night of February 27 she again committed the same imprudence, and it was after this that the symptoms, which were already imminent the preceding day, manifested themselves with awful violence.

She looked on the point of choking, and cerebral congestion

was also to be dreaded. She was immediately bled from the arm, but four hours afterwards Dr. Blondeau was sent for; the contractions had diminished in the limbs, but the symptoms had become worse in regard to respiration. The muscles of the neck and face were more violently contracted than in the morning; the livid face, the fixed eyes, the anxious breathing (which had already become stertorous), the uncountable pulse, pointed to asphyxia carried to the highest degree, and to imminent death; and yet, in the midst of this storm, the patient seemed to retain her consciousness. Twelve leeches were ordered to be applied behind the ears, but two or three had scarcely taken before the patient died.

On making a post-mortem examination, all the viscera were examined with the greatest care, and no other appreciable material lesion was found than traces of congestion in the meninges, the veins of which contained a little more dark blood than usual.

This is the only instance, gentlemen, in which I have known idiopathic contractions terminate in death, for the young man whom you saw in one of my wards, and who subsequently died under Dr. Rostan's care, died of tubercular consumption and not of his convulsive affection.

Notwithstanding the fatal case which I have just related to you, the *prognosis* of tetany is not grave. Even in its most severe forms, when the symptoms have sometimes become apparently serious, and have excited fears that death might occur, I have never seen a single patient die, and I have by this time seen a very considerable number of such cases.

After the complaint has lasted a variable period, from several days to one, two, or three months, the patient gets well, even when he has not undergone treatment; and the attacks, which leave behind only lumbago and transient weakness, do not seem to affect the system deeply, or to impair the general health.

Pathological anatomy has necessarily done very little in this complaint; but, from a mere review of the symptoms, it is impossible to admit that such mobile and transitory phenomena can be due to the existence of serious organic lesions. Those which some authors, Dr. Imbert-Gourbeyre among others, have regarded as the causes of idiopathic contractions, belonged to the diseases of which the individuals died, and in the course of which the convulsions had developed themselves. Some degree of cerebral congestion was, it is true, found in the woman who died under Mr. Cullerier's care; but it was an effect not the cause of the convulsions, or rather of the asphyxia, which had brought on death. In the case of the young man who had been in my wards, the softening of the spinal cord was itself a secondary change analogous to those met with in convulsive

diseases—facts to which I have sufficiently called your attention in connection with epilepsy.

Intermittent contractions have, therefore, been justly classed with neuroses, and regarded as a convulsive neurosis, like epilepsy, eclampsia, and hysteria, although we know less of their *nature*. Yet the conditions which are favourable for their development, the evident influence of cold on their production, the suddenness with which the symptoms that characterize them come on, the mobile and flying character of these, the intermission between the attacks, induce me to believe that they are of a *rheumatic nature*. In support of this view, in which they concur, several medical men have pointed out the coincident existence of rheumatism, and have insisted, as I have done myself, on the presence of a buffy coat on the blood drawn in such cases. This last argument, however, is perhaps of less value than we have ascribed to it.

I shall not dwell on the *differential diagnosis* between tetany and other forms of contractions, because, from what you have seen yourselves and what I have told you, it seems to me difficult to confound this complaint with any other. In the grave form alone, and the form of medium intensity, one may be for a moment in doubt, because he may, at first sight, think that the case is one of *idiopathic tetanus*. But while in this latter affection, the convulsions, whether they be regularly tonic or mixed up with clonic convulsions which preceded them, begin first in the muscles of the jaws, those of the face (producing trismus), and those of the trunk, and only by degrees extend to the extremities simultaneously, rheumatic contractions run an opposite course. It rarely happens, moreover, that the muscles of the extremities and those of the rest of the body are affected at the same time; lastly, the circumstance that it may be provoked by compressing the limbs is an important character, pathognomonic of the complaint.

I shall not speak of the differential diagnosis between tetany and contractions depending on cerebral or spinal diseases, the analogies between these being only very remote. Besides, symptomatic contractions are generally limited to a certain number of muscles exclusively, and are preceded or accompanied by a group of phenomena,—such as disorders of the intellect, impairment of sensibility, persistent paralysis, and febrile symptoms,—which essentially differ from what we observe in tetany, in which local manifestations, having themselves very special characters, are everything.

If you recall to mind what I told you of epilepsy, either in its convulsive or its partial form, you will understand why I do not dwell on the diagnosis between it and tetany, for, to my mind, it is attended with no difficulty. The retention of the

intellectual faculties in cases of general contractions, carried to the highest degree, is of itself sufficient to enable one at first sight to recognize them from an epileptic fit, and doubt is possible in such cases only.

Now what should the *treatment* of tetany be?

Bloodletting seemed to me from the beginning to be formally indicated, with the view of combating the congestion, which considerably alarmed me in the first cases which came under my notice. Although my premisses were wrong, I was thus led to apply a method of treatment which even now renders me the greatest services. Chiefly when the patient is of a vigorous constitution, and when there is very marked febrile reaction, I have recourse to *bleeding from the arm*, and to cupping of the spine. Whatever be the mode of action of this plan of treatment, its good results cannot be called in question, and I cannot be suspected when I advocate it, for you are aware that there are few men who are as chary of bleeding as I am. When I afterwards thought that this neurosis was of a rheumatic nature, I administered *quinine*, which is acknowledged by most practitioners to be efficacious in the treatment of rheumatism. Although I obtained some really good results from it, they were not, however, to be compared with those of bloodletting.

But there are cases in which bleeding is not admissible, and treatment must then consist chiefly in the administration of quinine. Thus, when the patient suffering from idiopathic contractions is of a weakly constitution, or has been debilitated by a chronic diarrhœa (as was the case in the woman whom you saw at No. 20 in St. Bernard Ward, and who nursed twins, of whom she had been delivered only a month before), bleeding would be productive of fatal consequences, so that the intercurrent accidents should be combated, the exhausting discharge arrested by all means, and, as soon as the stomach can bear it, quinine is to be given.

Opium and belladonna, in small doses, are useful adjuncts of bloodletting or of quinine.

In the grave form of the disease, and in violent paroxysms, as those of the woman in Lourcine Hospital, *chloroform inhalations* (practised with all the prudence demanded by such a potent drug and by peculiar idiosyncrasies) are indicated, as in convulsions in general. The young man who was in St. Agnes Ward begged for them himself during his fits, so great were his hopes of obtaining relief from it; and however temporarily it might be, yet chloroform never failed to relieve him. Lastly, gentlemen, you will find in the *Bulletin thérapeutique* for March 1860, a case reported by my regretted friend, Dr. Aran, of idiopathic contractions of the extremities cured by *local applications of chloroform* to the contracted muscles, simultaneously

with its internal administration, in doses of four or five minims every hour, in acacia mixture.

Aran appends to his case certain remarks which should be borne in mind. It should not be forgotten, he says, that chloroform irritates the skin very much; and, consequently, too large a quantity of the fluid should not be used in persons with a fine and delicate skin. A piece of fine linen impregnated with chloroform is alone required, and it is not even necessary that the whole piece be moistened, but only the part which is in contact with the contracted muscles. He also suggests that in women with a very fine and delicate skin, some advantages might be gained by using chloroform mixed with an equal or double the quantity of oil of sweet almonds, or of camphorated chamomile-oil. At all events, the piece of linen should be kept *in situ* by means of a few turns of a bandage, so as to make sure that the affected parts are in contact with the chloroform.

LECTURE XIV.

ON CHOREA.

Gentlemen,—Those among you who have for some years continuously attended my clinical lectures have seen, in our male and female wards, patients suffering from convulsive affections, characterized, all of them, by muscular agitation, more or less disorderly and strange movements and contortions, and to which the generic term *chorea* (from the Greek word χορεια, a dance) might perfectly be applied.

Quite recently we had three women, at the same time, in St. Bernard Ward (one, 21 years old, at No. 2; another, a young girl, 16 years of age, at No. 30; and a third, aged 19, at No. 31A), who were all suffering from the same complaint, which had, however, set in under very different circumstances; while these very same symptoms were present in a young man, 19 years old, lying in bed No. 4, St. Agnes Ward. You were, at first sight, struck with the look of hebetude and imbecility of this young man. He was constantly making grimaces, grinned for the least thing, and answered badly the questions put to him, seeming scarcely to understand them. The impairment of his intellectual faculties was, however, much more apparent than real; for it was the constant convulsive agitation of the muscles of his face which gave him that imbecile look, and the grimacing and grinning aspect which immediately attracted your attention. He kept also making disorderly movements, strange contortions, which were most marked in the extremities, chiefly in the arms.

At Nos. 8 and 9 in the same ward you could see two men—one aged 51, a hatter, in whose trade the acid nitrate of mercury is used for milling the felt of which hats are made; the other a house-painter, who affirmed that he had never had painter's colic or any other symptom of lead-poisoning, but who confessed that he was in the habit of drinking brandy every morning fasting, in quantity not sufficient to make him drunk, but, according to his own expression, sufficient to excite him. Both these men were affected with general trembling of the upper and lower limbs, which was so great in the case of the patient at No. 9 that he could not stand, even by holding on to his bedpost; he could scarcely eat, from the difficulty he had in carrying his food to his mouth, and he spoke with the greatest difficulty, from his tongue itself being involved in the disease.

Again, in bed No. 6, St. Bernard Ward, there was a girl 13½ years old, who was likewise affected with a convulsive agitation, which had manifested itself subsequently to accidents, of which she gave the following account. She enjoyed good health; she had menstruated, for the first time, eighteen months previously; and there had been no irregularity of this function, which had been at once established properly. She had never had hysterical fits, although she laughed and cried without a real reason, was frightened about nothing, and presented all the characters of a nervous mobile temperament. About sixteen months before the complaint set in for which she came into hospital, she had had typhoid fever of six weeks' duration, which had left behind it constant headache, to which she had not been previously subject. Five weeks before her admission she went to work as usual, but during the course of the day she was seized with convulsive movements of the arms and legs, which were after all rather moderate, for she went on with her work. The movements became more violent the next day, and were accompanied by other phenomena. She had, without cause, paroxysms of wild joy, which did not calm down even on her visiting her sick mother, and although she was deeply moved; on the contrary, her demonstrations of joy became more and more wild in the course of the day. She went to work again the next day, and kept on working as usual, in spite of the constant agitation of her arms and hands; but about 11 o'clock in the morning more serious symptoms manifested themselves. She turned suddenly pale, and nearly lost consciousness. As this state lasted, she was taken home; she then complained of shivering, and a general sensation of cold, which made her shake all over and her teeth chatter. She had a complete syncope at 4 P.M., soon followed by convulsions violent enough to require a strait-waistcoat to restrain her. This attack lasted an hour and a half, during which time she was delirious, alternately singing, and crying out fiercely, and frightening all the people in the house. Her face was red and swollen, and she looked haggard. She was then brought to the Hôtel-Dieu, where the attack quickly subsided spontaneously, leaving after it the convulsive agitation of the muscles which we saw.

As the patient lay on her bed, these convulsions consisted in alternate movements of flexion and extension, recurring continuously, and always in the same direction. If she were asked to take hold of an object shown to her, even when of small size (as a pin, for instance), she contrived to do it, moving her hand towards it in jerks, but without difficulty, and in a straight line. She never dropped the pin when she had once seized it; and although she continued to shake, she could fix it in her

dress. She could feed herself also, and easily guided the spoon from her plate to her mouth; whilst the man at No. 9 in St. Agnes Ward could not do this, nor the young man at No. 4, nor the three patients at 2, 30, and 31A in St. Bernard Ward.

The first thing which unquestionably struck you, gentlemen, in all these cases, was the presence of *choreic* movements; but before enquiring into other elements of diagnosis, apart from this common character, a moment's attention already enabled you to catch such distinct differences in the muscular agitation and the involuntary movements which characterised it, that you could not only recognise well-defined species belonging to the genus, but were also led to conclude that some of the species belonged to very different pathological genera.

Thus, while the young women lying respectively in beds 2, 30, and 31A in St. Bernard Ward, and the young man in bed 4, St. Agnes Ward, were affected with that kind of chorea which, since Sydenham, has been called *St. Vitus's dance*, the young girl at No. 6, in the female ward, suffered from an hysterical choreiform affection; the two men at 8 and 9 in St. Agnes Ward had, the one alcoholic chorea, and the other mercurial chorea—or, if you like, *trembling*, a term applied to those species of chorea in nosological tables.

I have been accused of changing the meaning of the word *chorea*, admitted by everyone, it is said, to designate what I call, after Sydenham, St. Vitus's dance, and of thus confounding with chorea, properly so called, various choreic affections, such as tarentismus, hysterical dansomania, hysterical chorea, and trembling, which nobody ever thought of mixing up together. My answer is, that I am not the only one who has taken in its widest acceptation an essentially generic term. As to the confusion which is laid to my charge, I will endeavour to put you on your guard against it (precisely because I too often see medical men commit that error), if not by describing to you all the species of chorea, some of which besides, such as the epidemic choreomania of the Middle Ages, and tarentismus, are almost unknown now, but by passing some of them at least in review, and especially by speaking to you of St. Vitus's dance, to as great a length as the nature of this course admits, and by pointing out the characters which distinguish it from other choreiform affections.

ST. VITUS'S DANCE.

(CHOREA SANCTI VITI, SYDENHAM.)

Reason why the term St. Vitus's Dance appears to me better than that of *Chorea*.—Predisposing causes: age, sex, hereditary influence.—Pathological conditions: Chlorosis, Tubercular and Strumous Diathesis, Rheumatism.—Exciting causes: emotions, fright.—Description of the disease.—Antecedent phenomena.—Convulsive phenomena.—Their specific character.—Paralysis.—Disorders of Sensibility.—Impairment of the intellectual faculties.—The complaint is usually curable.—Its mean duration.—It may terminate in death, and how.—Pathological anatomy throws no light on it.—Influence of Intercurrent Febrile Diseases on the course of the complaint.—Relapses and recurrences: their duration is less than that of the previous attacks.—Treatment: cold and warm baths, sulphur baths, gymnastics.—Internal remedies: tartar-emetic, strychnine, opium in large doses in grave cases, hygienic measures.

GENTLEMEN,—It is beyond question, especially since the beautiful historical researches made on this point by Messrs. G. Sée and Roth,[1] and some others, that the name St. Vitus's dance was at first given to a singular disease, very different from the one which we now know, and which was epidemic in several German villages at the end of the fourteenth and the beginning of the fifteenth century. This name was applied to it because individuals suffering from this choreomania—a regular extatic frenzy, to which that of the convulsionists at St. Médard has been compared—went on a pilgrimage to St. Vitus's Chapel, at Dresselhausen, in the district of Ulm, in Suabia, as the saint was said to have the power of curing them, just as in our own time other saints are said, in popular legends, to possess an analogous power in other complaints. Whatever its origin, the name St. Vitus's dance, perverted from its original meaning, was given by Sydenham, who did not pique himself on being an erudite, to the complaints of which I am now speaking; and from having been adopted by the authors of the eighteenth century, it is now understood by everyone.

It has been adopted in your very textbooks, and there given as synonymous of *chorea*, a generic term which Bouteille proposed, in 1810, to substitute for it; while it has taken such firm root in medical language, that all efforts made to restore its first meaning to it have failed.

If this is to be regretted, in an historical point of view, science and practice, I must hasten to add, do not lose much by Sydenham's mistake in erudition; for it is to this great physician that we are indebted for the first truly scientific

[1] Germain Sée, "De la chorée" (Mém. de l'Acad. de Méd. 1850, t. xv. p. 373). Roth, "Histoire de la Musculation irrésistible" (Paris, 1850).

description of the symptoms of the complaint. For my part, now that everybody understands what is meant by *St. Vitus's dance*, this name appears to me the best—better than that of *chorea*, which, in its generic acceptation, includes many things and specifies none, while the former term applies to one complaint alone and to the whole of it, as the rule is in sound logic. It has the immense advantage of designating the disease in question better than could be done by any other name constructed according to the principles of the most correct nosology: whatever be the ideas entertained of the nature of the disease, this name prejudges nothing, and all theories can conveniently adapt themselves to it. The same thing happens in the case of this word as in that of all those words which mean nothing in themselves but have been adopted by custom: they are the best, because they include a complete definition, and convey to the mind a complete idea of the object meant. So it is with the terms *coqueluche* (whooping-cough) and *vérole* (pox), for instance, which, in spite and perhaps on account of their strange etymology, have become part of ordinary as well as of medical language, and which could not be replaced by any other term borrowed from a nomenclature having high pretensions to scientific accuracy.

I now pass on to the study of St. Vitus's dance.

Although a certain number of cases of this complaint may be every year seen in my wards where patients over 16 years old are alone admitted, such cases are rare when compared with those that are met with in children's hospitals; and I shall only tell you what you know already, when I say, that *St. Vitus's dance* is a complaint occurring in *childhood* and *puberty*, and generally from 6 to 15 years of age. It is only exceptionally that it affects children before they have changed their first teeth,[1] and it is much more common to see it in individuals who have attained the age of puberty, up to 25 years. There are even instances of chorea on record attacking older persons; and M. G. Sée has seen it in a woman 36 years old, in another 44 years of age, and in a man aged 59. Jeffreys saw it in a patient 60 years old, and Powel and Maton in another 70 years old, while Bouteille saw a man aged 72 who was afflicted with it; and lastly, only recently, Dr. Henri Roger has recorded a case of chorea occurring in a lady 83 years of age.

This last case is so interesting, on account of its singularity, that I must beg permission to read it to you in full:—

"Mrs. ***, 83 years old, has as strong a constitution and as

[1 I have at present under my care a little girl, of strumous habit and puny make, who is only six years old, and is suffering from a most obstinate attack of chorea. She has not yet cut a single permanent tooth.—Ed.]

clear a mind as may be expected at her advanced age. Apart from some weakness in the legs and palpitations of the heart, of which she has complained for about the last ten years, unaccompanied by murmur and by marked præcordial dulness, and without a history of antecedent acute articular rheumatism; except also a rather obstinate constipation, and some vague rheumatic pains in the loins and the limbs, her health is at present as satisfactory as possible. I must mention, however, that eight years ago, I attended Mrs. *** for an attack of pleurisy with effusion on the right side; and two years ago for sciatica, which was of moderate intensity and duration; and last year for cerebral congestion, which disappeared after a few days.

"On the 15th of May last, I was sent for to see Mrs. *** and easily recognised chorea. For three or four days previously only, she had felt, without any appreciable cause, any intense emotion, or other premonitory symptoms, some uncertainty and exaggeration in the movements of her right arm and leg. These two limbs, when I saw her, were the seats of other marked movements: the arm was, at very short intervals, moved suddenly and with a jerk; when, in obedience to the patient's will, it was drawn forwards, it was soon pulled backwards or dragged more forwards by involuntary contractions; its movements were strange, irregular, and badly coordinated. The same thing occurred with the leg, which, although it lay on the bed, was drawn up by a sudden contraction, in such a manner that the foot was thrust at haphazard in various directions.

"When asked to do so, the patient could, by an effort of the will, stop these movements, but they began again almost immediately. Their uncertainty and irregularity increased still more when the patient was up. She could scarcely keep on her legs, and was compelled to sit down instantly; with some care, and with time, she managed to feed herself. The face was only slightly distorted, from the muscles of the face being less frequently and less violently contracted than those of the limbs. Speech was nearly natural, and was interrupted at rare intervals only.

"The muscles of the walls of the chest and abdomen were not the seats of special contractions. The senses were not markedly affected. The patient complained of a sense of fatigue all over, owing to the exaggerated motility. General sensibility was neither diminished nor exalted; Mrs. * * * was low-spirited, or rather had grown impatient, on account chiefly of her sleeplessness, although she had some sleep, during which the chorea ceased. The animal functions (digestion, circulation, urinary secretion) were normally performed. These details sufficiently prove the existence of idiopathic chorea: the disease, let it be added, was at first moderately intense, but

increased in violence after three or four days. The movements became more incessant and more violent, were almost more marked in the arm and leg, and exclusively limited to the right side. The patient could not feed herself, and was not able to walk, while the chorea persisted during nearly the whole night, and prevented sleep. The disease continued in this degree until June 1, that is to say, for about a fortnight. From that date it decreased by degrees, and on June 15, namely after five weeks, the patient got perfectly well. During the whole time there was no impairment of the general health, and there was no concomitant phenomenon deserving of notice, except the coexistence of neuralgic pain along the course of the arm, on a level with the insertion of the deltoid and about the elbow (without swelling or redness of the parts, and without fever).

"The treatment was simple. It consisted in the internal administration of oxide of zinc and of powdered belladonna, in gradually increasing doses of from 5 to 15 grains of the former, and from 1 to 2 grains of the latter. Local applications of chloroform diluted with water (1 part to 30) were used, and calmed the pain in the arm, which was the seat of the choreic movements, and the limbs were rubbed and neaded, especially the leg, which was not painful."

An analogous case is reported in Graves's "Clinical Lectures." The chorea was very violent, and the patient was a Dublin chemist, 70 years of age.

Dr. Henri Roger justly remarks that his patient was really suffering from St. Vitus's dance. "The complete integrity of the functions of the nervous system before the setting-in of the convulsive affection, the absence of all antecedent or subsequent cerebro-spinal disease, the unequivocal form of the symptoms (which were choreic and not choreiform), the duration of the neurosis, which was almost the usual one in such cases, and its favourable termination," amply justify the diagnosis.

These rare instances of St. Vitus's dance affecting individuals after puberty have almost exclusively occurred in women. *Sex*, therefore, plays in such cases a very important part as a predisposing cause; and this influence of the female sex is very remarkable at the periods of life in which chorea most generally manifests itself, for statistics show that the proportion between girls and boys is as 3 to 1. This proportion is still higher after puberty, and it may be stated that St. Vitus's dance occurs exceptionally in males after the age of 15, while a pretty good number of cases of the disease in females might be cited. While on this point I must call your attention, gentlemen, to the fact that articular rheumatism attacks males more frequently than females, and that this tends to detract from

the value of the opinion which holds that St. Vitus's dance is an expression of the rheumatic diathesis.

I need not mention, as some authors have done, that the nervous temperament more than any other predisposes to this neurosis. Dr. G. Sée has done away with this commonplace remark, as well as with the influence of the patient's constitution.

The same cannot be said of *hereditary predisposition*, which is unquestionable; and even if judicious statistics had not proved it, it might have been asked why St. Vitus's dance should not be subjected to the same law as all nervous diseases in which hereditary predisposition holds such an important place.

On enquiring into the family history of individuals affected with chorea, you will find that either their direct or collateral ancestors (of the latter, according to some, but for what reason I know not, no account should be taken) have suffered from various neuroses, such as hysteria, epilepsy, or eclampsia; or you will ascertain the existence of certain diathetic manifestations, and in particular of the tubercular diathesis. And here, again, we have to deal with the question of the mutual transformation of diatheses, a great question of general pathology, to which I have already alluded several times.

Several pathological conditions have been regarded as predisposing causes of chorea, but their influence for the most part is far from being proved. I do not stop to speak of the metastases of eczematous diseases—of the itch, of febrile eruptions, and of the metastases sequential to the suppression of habitual discharges—which have been too often erroneously enumerated among the etiological conditions of diseases the real cause of which escapes us; nor shall I say anything of gastro-intestinal disorders and of intestinal worms, which stand to chorea in a very doubtful relation of cause and effect, however marked their influence may be on the development of eclampsia. I will add that the impairment of the digestive functions—which, as I shall tell you presently, is a very common complication of St. Vitus's dance—is a consequence of the perturbation of the whole nervous system arising from the disease itself, and should not be regarded as its starting-point. It is unquestionable that St. Vitus's dance has often a marked influence on the development of *chlorosis*. It is equally unquestionable that a large number, the largest number even, of choreic women were previously chlorotic, and that they get well when they are cured of chlorosis by the measures indicated in such cases. It is evident that chlorosis is very often a concomitant condition, at the very least, which should be taken into account in the treatment of St. Vitus's dance. I will go further: chlorosis, like all causes capable of weakening the organism and of producing erethism of the nervous system, plays an important part in the

etiology of this singular neurosis. In *pregnancy*, which may be regarded as a favourable condition for the production of this nervous complaint, the latter should be ascribed to the chlorosis which so frequently accompanies pregnancy. No one denies this indirect influence of pregnancy on the production of St. Vitus's dance. Dr. G. Sée has collected sixteen instances of the disease occurring in women from 19 to 20 years of age, and my colleague, Dr. Horteloup, has seen one case in a young woman aged 16.

I told you a moment ago, gentlemen, *à propos* of hereditary predisposition, that St. Vitus's dance could be the manifestation of certain diatheses which had shown themselves in the direct or collateral ancestors of the patient, in their usual form. I would not go so far as to say, with J. Frank and Dr. G. Sée, that the tubercular or strumous diathesis plays an important part in the production of chorea, although a large proportion of choreic patients are also tubercular. The proportion which exists between other chronic diseases and tubercles should be first established.

But of all these predisposing pathological states, rheumatism is assuredly the most marked and the least questionable. The relation of *rheumatism* to *St. Vitus's dance* had been partially seen by Stoll, by Copland, by Bouteille, by Abercrombie, Begbie, Bright, Gabb, and Richard; while others, again, had pointed out the coexistence of pericarditis and endocarditis with chorea. Dr. Botrel went further in 1850, when he chose for the subject of his thesis, *Of Chorea considered as a Rheumatic Affection*, and propounded the opinion, professed before him by Dr. Hughes, that the former complaint was only a special manifestation of the latter. But in his remarkable memoir on *Chorea and the Nervous Affections*, &c., which, in 1851, gained a prize at the Academy of Medicine, Dr. G. Sée has brought out this fact so prominently, that the greater portion of this discovery really belongs to him.

The interesting researches made by Dr. Sée, who is physician to the Hôpital des Enfants, led him to the conclusion that in nearly every case of St. Vitus's dance, rheumatic pain had at least been complained of. Dr. Sée has not, however, guarded himself from exaggeration, and has confounded under the same head, Rheumatic Affections, simple lumbago and muscular pain, which so frequently accompany the invasion of chorea.

This law, however, when made less exclusive, is an acquired fact in science, and there is no practitioner nowadays who has not been able to verify it. On several occasions, I showed you how it applied to cases that we saw together—among others to the case of a poor young woman in St. Bernard Ward, who was

carried off by a most violent attack of chorea, which manifested itself ten or fifteen days after the setting-in of acute articular rheumatism.

About the same period I was asked by my colleague and friend Legroux to see with him the daughter of a tailor in the *Rue Richelieu*, who was suffering from an attack of acute and general articular rheumatism. We found endocarditis also; and the pain persisting ten or fifteen days after the outset of the rheumatic fever, St. Vitus's dance set in. It was moderate at first, but soon became complicated with awful muscular disorders, delirium, and lastly comatose symptoms: the girl died on the seventeenth day.

Dr. E. Moynier published, in the thesis which he wrote for his doctor's degree in 1855, the following case, which I had communicated to him. A girl, 10½ years old, has a first attack of chorea, after which she becomes hemiplegic. At the age of 14 she has rheumatic fever, and subsequently a second but slight attack of St. Vitus's dance. Her brother, when 13 years old, had had rheumatic fever, and two months afterwards had been seized with the same convulsive affection as his sister. Their father had on five several occasions suffered from articular rheumatism, but never from chorea.

A boy, 5½ years old, is seized on January 1, 1859, with articular rheumatism, which lasts a month. On the 1st of February following he had St. Vitus's dance, which was still present on March 7, when I saw him, and I recognised endocarditis, characterized by a rough cardiac murmur.

I could add a good many more cases which have come under my own observation, and some of which are quite recent; for I never allow the opportunity to pass now, of enquiring into the law of coincidence, to which the labours of Drs. Hughes, Botrel, and G. Sée have called my attention particularly. Profiting by their researches, I have in many cases been able to foretell that children suffering from rheumatism would become affected with chorea. On the other hand, I have been able to predict that choreic children who were brought to me would, sooner or later, have rheumatism. Yet you will rarely see it precede rheumatism, while it often follows it, in the proportion of one-third of the cases.

This proportion, which is nearly the one given by Dr. G. Sée, may perhaps seem exaggerated, if cases of purely articular rheumatism be alone reckoned; but the great pathological law laid down by my eminent colleague at the Charité Hospital, Dr. Bouillaud, namely, the law of coincidence between cardiac affection and rheumatism, comes here to our help. For, if you do not find articular rheumatism in a pretty large number of choreic patients, you will find the signs of old endocarditis,

a manifestation of rheumatism which spared the joints, but existed nevertheless, and affected the organism deeply. Allow me to relate to you a case which you saw with me.

A girl 14 years old, who had never menstruated, was admitted into the clinical wards on January 9, 1861, for St. Vitus's dance, affecting chiefly the left side. She had been ill for twelve days, and gave us very incomplete information as to her previous history. I found out, however, that when a child she had had choreic movements and articular pains. Her face wore a very marked expression of hebetude; she could scarcely speak, and her lips moved in a singular manner. When she tried to speak, she protruded her tongue out of her mouth in jerks; and when she drank, she swallowed the liquid spasmodically. She could scarcely walk; her left arm and hand and her left leg were shaken in a disorderly manner; she was obliged to keep in bed, and could not feed herself. Sensation was diminished on the left side, both in the face and limbs. There was no intestinal disorder; respiration and circulation were normal; but over the cardiac region, especially at the apex, there was heard a soft systolic blowing murmur, which did not extend into the bloodvessels.

On January 16 she had on the limbs velvety eminences, like those of urticaria; on the 17th she had fever, characterized by a frequent pulse and heat of skin. She complained of rheumatic pain in several joints, and there was marked effusion into the right knee. The cardiac blowing murmur was more distinctly heard, and more prolonged.

For seven days, several articulations were attacked with rheumatism, which left them to return again after a time, and meanwhile the choreic movements nearly disappeared. On January 25 the aspect of stupor was very marked; the expression of the face never varied, and the pupils were dilated. The child lay on her back, scarcely complaining of pain in the joints, and she had convergent strabismus. There was, however, marked diminution of the pulse and of the respiratory movements. Since January 20 the digitalis had been stopped, which had been administered for several days, without producing any perceptible modification of the heart's pulsations, which until then had been frequent, and been felt over a broad area, as they usually are in rheumatism. The frontal headache, the strabismus, stupor, diminution of the movements of the heart and of respiration, the nearly complete cessation of the pain in the joints, were sufficient indications that rheumatism had attacked the brain; yet the cerebral macula only became manifest on January 26, but very conspicuously. There was constipation also. The brain-symptoms persisted during fourteen days, the pulsations of the heart had become

less and less frequent (48 per minute), and the breathing was slow, sometimes interrupted for a few seconds. Four ounces of coffee a day had at first been given, and subsequently calomel, in divided doses. On February 4, that is, fourteen or fifteen days after the onset of the cerebral rheumatism, all the brain-symptoms improved: there was less stupor, the intellect was clearer, the strabismus less marked, the pupils less dilated, and the patient answered questions readily, while she had not been able to do this for several days. The pulse became more frequent, and the respiration more regular; the face no longer had the same bluish tint, and the cerebral macula was less developed, and lasted a shorter time. From that time the improvement increased every day, markedly and continuously, and all the brain-symptoms disappeared soon, and pain was no longer complained of in the joints. As the appetite had returned, nourishment could be given. The girl was fairly convalescent, although her face still wore a singular expression; and although she had no choreic convulsions, her voluntary movements were still slightly uncertain. The cure was afterwards complete.

In this case, gentlemen, St. Vitus's dance opened the scene; acute articular rheumatism soon followed, preceded by cardiac symptoms, and then there supervened a grave complication, cerebral rheumatism. Rheumatism attacks children more frequently than is believed. Independently of the causes which produce it in adults, and to which children are equally liable, there is one cause to which they are more exposed than others, namely, scarlatina. When I come to speak of this exanthematous fever, I shall dwell fully on the coincidence of rheumatism and scarlatina; and I will tell you that it is pretty common (less so in children, however, than in adults, in whom this occurs in one-third of the cases) to see rheumatic affections set in during the acute stage of the eruptive fever; but as the rheumatism does not give rise to the general symptoms which usually characterize it, as the patients complain little of it, and as it is most frequently confined to three or four joints (chiefly the wrists), it is often overlooked. Yet by carefully questioning the patients, by examining their joints with attention, and slightly compressing them, pain is found to exist in the joints from the third to the eighth day of the disease, sometimes later. Thus is explained the production of endocarditis and pericarditis, complications which manifest themselves when scarlatina is declining, pericarditis somewhat more rarely than endocarditis.

Deep emotion, from any cause, and most particularly *fright*, is a *determining cause* of St. Vitus's dance. The young girl, 16 years old, who lay in bed 30, St. Bernard Ward, afforded an instance of this. Her previous health had always been

good; she had never had rheumatic pains (and careful auscultation detected no sign of cardiac disease), and her complaint dated a fortnight back. A man caught hold of her one evening as she was going downstairs without a light, and she was so frightened that she had a nervous fit, and from that moment became affected with St. Vitus's dance. The disease was developed to a pretty high degree, and her case could be regarded as typical.

Several among you may recollect another girl, aged 17, who was sent into my ward by Professor Jobert, in December 1860. She had an artificial anus in the umbilical region, which had rendered a surgical operation necessary. She had always been very nervous, and had a strange temper; and she was so alarmed by the operation, that she was immediately seized with St. Vitus's dance, which was very grave, was attended with delirium, and got well by slow degrees also.

The *invasion* of St. Vitus's dance is rarely sudden as it was in these two instances; in the immense majority of cases there are *premonitory phenomena*, which often escape notice, and thus induce the belief that the choreic movements developed themselves at once.

These prodromata consist in impairment of the *intellectual faculties*. The child's temper changes; the joyousness habitual to its age is replaced by unusual sadness and morosity, and he becomes capricious and agitated; he sheds tears copiously for the least thing; he is irritable; his natural timidity grows worse; he seeks solitude, and keeps away from his playfellows. He becomes at the same time incapable of fixing his attention long; his aptitude for work diminishes; his memory is less retentive; and this enfeeblement of the intellect, which does not escape the attention of mothers (who, however, are always ready to exaggerate their children's qualities), increases still more in proportion as the disease progresses. I will presently revert to this important point.

Generally, also, the patient complains of malaise, of headache, of vague pains in the limbs, and of præcordial anxiety. The digestive functions lose their accustomed regularity; the appetite diminishes, digestion becomes more difficult, and there is constipation. The convulsive agitation is already announced by a wish to move constantly from place to place, and by uneasiness in the limbs; this agitation becomes more and more marked, and, lastly, the choreic convulsions manifest themselves.

The *symptoms* of the confirmed disease show themselves sometimes in the upper, at other times in the lower extremities, and at others again in both at the same time. In some cases the face gets distorted first, but more frequently the upper limbs

are the first to be affected, and it is of very rare occurrence indeed that the disease is general from the outset.

As a rule, I repeat, chorea begins in one side and attacks the other side by degrees, involving the trunk and face also. In some very rare cases it is localized during the whole course of the disease, and we had in St. Bernard Ward an instance of this *hemichorea*. The right side was affected in that case, while most commonly unilateral chorea is on the left side. Even when general, chorea always presents something of a unilateral character—that is, the convulsive movements are more marked on one side than on the other, more particularly on the left. This may take place alternately also; for instance, the agitation may cease on the side which was most affected, and may become more violent on the other.

Chorea which is *partial* at the beginning may remain so throughout, or after becoming general it may afterwards affect a few muscles only. Such cases are rare, however, and a great many of those which have been reported as such were not instances of St. Vitus's dance, but of *tic*, a species of chorea which should not be mistaken for it.

If, at the outset, the symptoms which characterize this complaint are sufficiently slight not to attract the attention of the child's friends, and if they then consist merely in a want of precision of the voluntary movements, or in a sort of carphology, or in some more or less transient contortions of the trunk and face; when the disease is fully developed, it can no longer be mistaken, and the most minute description cannot give an accurate idea of its strange and varied aspect.

One is struck at first sight with the singularity, the uncertainty, and irregularity of the child's movements. He cannot remain a single moment at rest. He has a difficulty in remaining in the standing posture, for his legs bend under him, and then straighten themselves in an instant; his gait is peculiar, and he runs rather than walks. If he tries to take a step forward, he raises his foot higher than he desires, thrusts it right and left; and scarcely has this foot touched the ground again, than the other gets off at once and moves in a similar manner. His walk consists in constant leaping—in a sort of ill-cadenced dance, which assumes a more grotesque character, painful to witness, from the irregular movements of the upper limbs, the contortions of the trunk and head, which, according to Dr. Rufz's comparison, make the poor choreic patient resemble one of those puppets that are moved by strings. When the symptoms are very severe, the standing posture and progression are perfectly impossible, and the patient is compelled to remain in bed under pain of falling down without being able to get up again.

The upper limbs move likewise in different directions. They pass, with excessive rapidity, from a state of flexion into one of extension, from pronation into supination, and these various movements succeed one another without regularity. The patient succeeds in reaching a determined spot with his hand only after many efforts. If he tries, for instance, to carry it to his head, he raises his arm up, after many false moves, striking his face and forehead while doing so, and he is unable to retain that position long. When he tries to take hold of any object presented to him, he thrusts his hand forward as if his arm moved by means of a spring, then withdraws it with the same suddenness, without reaching his aim or going beyond it, and attaining it at last after numerous attempts; even when he gets at what he desires, it often is by upsetting it, and throwing it away from him. After seizing it he is on the point of dropping it suddenly; and when he has got hold of it at last, if it be a glass, for instance, and he tries to drink, he only succeeds with great difficulty; and before he does so, as Sydenham says, he makes a thousand-and-one contortions, moves his glass right and left, until, on its meeting his lips by chance, he gulps down the liquid; or, again, he holds the glass between his teeth, and lets it go only after emptying it. You may conceive, gentlemen, how difficult it is to nourish a patient in such cases, and why they have to be fed by others.

The face wears a singularly imbecile look from the convulsions of its muscles, which give rise to grimaces of the most varied kind. The eyebrows, the skin of the forehead, the alæ nasi contract and relax; while the eyelids are alternately raised and lowered, the lips pulled in various directions, the mouth opens and closes unceasingly, and the eyes roll convulsively in the orbit.

As the muscles of the tongue are involved as well as the rest, speech is often hesitating, or the patient actually stammers, and can be understood with difficulty. Articulation is all the more embarrassed that the muscles of the larynx are themselves involved in some cases, and the sound of the voice being then altered, the patient utters a kind of bark.

Strange sounds are occasionally produced through the voice coming out in inspiration instead of expiration. While the patient expires in the act of speaking, the inspiratory muscles suddenly contract convulsively, and cause the air to rush into the larynx; so that, from this kind of antagonism between the mind that wills the speech and the inspiratory muscles, the voice undergoes a strange alteration.

Lastly, the pharynx and other muscles of organic life may be affected; deglutition is then impaired, while, owing to the relaxation of the sphincters of the rectum and bladder, the urine

and fæces are passed involuntarily. Such cases, however, are somewhat rare.

Choreic convulsions, therefore, attack the muscles of the life of relation almost exclusively; and although the movements are involuntary, like all convulsions, the will still possesses a certain influence over them. The want of coordination seems to result from the fact that some of the contractions are involuntary and others voluntary, but the latter are not in sufficient number to neutralise the former. I will explain myself.

When the will commands freely—as, for instance, when it orders the arm to rise, or the leg to move forwards, the muscles which are charged with the execution of these movements do so with perfect regularity; they act with coordination, and in a perfectly harmonious order. Now, while this harmony persists still in hysterical chorea, and in the various kinds of trembling, in which the will is incapable of preventing the convulsions and yet commands combined movements, it does not obtain in St. Vitus's dance. In this complaint, on the contrary, it seems that the will is powerful enough to call the muscles into action, but is unable to direct or moderate them by means of the antagonistic muscles when the impulse has once been given; it seems that, instead of obeying then a single will, each muscle contracts at its own pleasure, or obeys different wills. This is an important fact, which is observed in St. Vitus's dance, and sometimes also in *locomotor ataxy*, as I have already told you.

There is another phenomenon which is likewise special to this kind of chorea, namely *paralysis*, which is almost always present. The limbs which are most affected with choreic movements are the seat of the paralysis; the arm, for instance, which is the most convulsed, is the one also in which muscular strength is most diminished. The child often complains that this arm is heavier than the other. The leg which is most convulsed is also the one which bears the weight of the body least, and which is dragged the most when the child walks. This coexistence of a greater degree of convulsive agitation, and of a diminution of muscular strength, is all the more inexplicable that the paralysis is as mobile as the choreic affection with which it is connected. Thus when the chorea is more marked in one half of the body, the paralysis is also marked on that side; but if the convulsions become more violent on the opposite side, that side will in its turn be paralysed.

This paralysis disappears almost always simultaneously with the chorea, but it may in some cases persist after it, and be complicated with atrophy of the paralysed muscles, constituting then a more or less durable infirmity. In some still rarer instances, paralysis (I do not mean a mere diminution of muscular

strength, but true paralysis) precedes the manifestation of convulsive phenomena.

A girl, 18 years old, was brought to Paris by her mother, who was alarmed at seeing her seized with right hemiplegia. Professor Andral and I were asked to see her, and we made out that, besides a marked diminution of muscular strength, there was also a very appreciable diminution of cutaneous sensibility on the right side. On carefully examining the patient, however, we noticed that her right foot was constantly adducted and abducted in turn, that her hand was also perpetually agitated, her fingers bending and then straightening themselves out. Moreover, the patient kept her head inclined on her chest, and her face wore a singular expression of sadness and of fear. We immediately thought of St. Vitus's dance, and asked the mother whether these movements had existed for a long time, but they had not yet attracted her notice. The characteristic symptoms which soon afterwards manifested themselves proved the accuracy of our diagnosis.

The diminution of sensibility which we found in this case exists in most instances, for *disorders of sensibility* are nearly constant in St. Vitus's dance. I have already spoken to you of the vague pains which the patient feels in his limbs, and which, after announcing the invasion of the disease, persist when it is fully developed. To them are then superadded a sensation of formication, of tingling, and more or less marked *anæsthesia*, which is always greater on the most convulsed side. You saw me prick and pinch the young woman in bed 31A, in St. Bernard Ward, and thus recognise this perversion of tactile sensibility. This patient also told us that she could not see very well with her right eye, and that this weakness of sight had set in since the first attack of the same complaint, which she had had a year before, and that it had never improved. This *impairment of sight*, which is probably due to paralysis of the retina, has been pointed out by several authors; Dr. G. Sée records an instance of it which fell under his own observation, but he justly adds that this accident is excessively rare.

The convulsions, and the motor and sensory paralysis, are not the only indications of the perturbation of the nervous system. With very rare exceptions, there is in every case a more or less marked *impairment of the intellectual faculties*. This consists in a deeper modification than the timidity and the change in the moral disposition of the patient which I have mentioned already. I do not mean, gentlemen, that a person who is afflicted with St. Vitus's dance becomes demented or an imbecile; but although he looks stupid, owing to the singular mobility of his features, and the impediment in his speech (which circumstance may certainly mislead, and induce the

belief that the intellectual impairment is greater than it really is), yet it is unquestionable that his intellect is below par. If he happens to be at school, the change which has taken place in him is found out by his losing his place in the class. In some exceptional cases there have been signs of real insanity, and you saw an instance of this in the young woman of whom I have already spoken, and who became choreic after an operation performed by M. Jobert.

This intellectual disturbance is as transitory as the disease itself. There are instances on record, however (rare though they be), of children who never again showed the same degree of intelligence as before they became affected with St. Vitus's dance; and cases have been even related in which deep changes had been left behind—namely, a certain degree of hebetude, and even of mental alienation.

It more frequently happens that nervous excitability and an exaggerated sensitiveness persist in some cases.

These disorders of innervation manifest themselves also in the organic functions, and to them are due the præcordial anxiety and the palpitations of the heart complained of by the patient. The latter are accompanied by a soft blowing murmur, which is heard over the base of the heart, along the vessels of the neck. It is an anæmic murmur, which should not be confounded with the rough bruit that characterizes rheumatic endocarditis, and it is owing to chlorosis, which often complicates if it does not precede chorea, and may be regarded as an effect of the influence of this disease on nutrition. The chlorosis is, besides, characterized by the discoloration of the integuments, by vertigo, headache, neuralgic pain, singing in the ears—sometimes by swelling of the face, and in girls by dysmenorrhœa and even amenorrhœa.

The disorders of the digestive functions, which showed themselves from the beginning, either continue, or reappear and produce gastralgia. There comes a time when the appetite, at first capricious, is lost entirely, when digestion is painful, and there is actual overloading of the stomach. Constipation is also habitually present, as Sydenham pointed out long ago.

Emotion increases the violence of the convulsions, and you should bear this in mind, lest you be mistaken as to the real gravity of a case, on seeing for the first time a patient who is not used to you.

It is a remarkable circumstance, which happens in every case, that these convulsive movements, however disorderly, violent, and persistent they may be when the patient is awake, cease completely during sleep, and the patient looks as quiet as in health. In grave cases, however, he is restless, his sleep is of short duration only, and interrupted by bad dreams. In still

more severe cases, the excessive agitation of the nervous system produces insomnia, which in its turn, acting as a cause of greater excitability, gives the unfortunate patient no rest at all. Brain-symptoms then set in—delirium, coma—and the patient gets into a state of exhaustion which tends to a fatal termination. When I come to speak of treatment, I shall tell you how to combat this dangerous complication, which, if not opposed in time, becomes so severe as to be soon beyond remedy; and I shall tell you also that, although they be of real and unquestionable utility, these measures are no longer useful, and should be replaced by others as soon as the disease resumes its regular course.

Although St. Vitus's dance usually *terminates* favourably, and gets well after having *lasted* from one to several months, it may not only leave behind it, as I have told you, excessive nervous irritability, partial paralysis, and intellectual debility, but it may also cause *death*. Although such cases are rare, they are but too frequent still; and I told you of two instances—one that of a young woman in St. Bernard Ward, and the other of a girl attended by Dr. Legroux and myself. I have myself met with five or six cases of the kind in the course of my practice, and M. Moynier has related several similar instances of the kind in his thesis.[1]

Death may take place from the extreme agitation; it may be due to nervous exhaustion, or to cerebral rheumatism (as in cases which I shall relate to you), or it may be the result of no less formidable accidents. The patient may die of a fever similar to that which kills persons who have been burnt over a large surface, and the analogy is the more striking that this fever arises from more or less numerous and extensive wounds, which are produced in the following manner.

I told you that choreic patients were sometimes unable to stand, and were compelled to remain in bed. Their agitation is then so excessive that they are kept in bed with the greatest difficulty. Their movements are so disorderly and violent that they knock themselves against the wood or ironwork of the bedstead, bruise themselves severely; and these bruises, getting inflamed, become the starting-points of purulent infiltrations and of phlegmonous erysipelas. Or, again, they rub off their skin, which they literally wear out by constantly rubbing against the bed-clothes, which they tear to pieces. Horrible wounds are thus produced, which, deepening by degrees, reach the bony prominences of the heels, the malleoli, elbows, spine, and scapulæ. You may conceive the consequences which must

[1 Three instances of chorea terminating in death are recorded by Dr. Chambers ("Lectures, chiefly Clinical," 4th ed. p. 369).—Ed.]

follow the pain and abundant suppuration to which these wounds give rise.

Such wounds are the more easily produced that the same thing happens in St. Vitus's dance as in grave fevers—in all diseases, in fact, which deeply affect the nervous system, and in which there is a marked tendency to suppuration and ulceration.

The following case, which was communicated to me by a country practitioner, is an important one, as bearing on this point :—

A young girl, whose mother was healthy, but whose father had been subject to eczematous eruptions, and who was of delicate health herself, and had had, in the preceding year, eczema of the head, neck, and shoulder, became choreic. The disease grew so violent in a few days that she was unable to feed herself. Strychnine was given, in gradually increasing doses (up to $1\frac{1}{5}$ grain) in the course of the twenty-four hours ; it did not produce tetanic rigidity, and quieted the symptoms markedly, so that the patient was soon able to drink by herself, with scarcely any difficulty. The tip of the right thumb, however, became affected with a whitlow, which got well rapidly ; but two days after the wound had healed, although the convulsions had notably improved, the child was seized with high fever and diarrhœa. Diffuse phlegmonous inflammation of the hand soon showed itself, which in less than twenty-four hours involved the back of the wrist and forearm, so that several incisions had to be made. From the outset the greatest precautions had been taken, in order to prevent the excoriations which the agitation made one dread. The patient was placed on a mattress laid on the floor, and constant watch was kept over her. Later, when the convulsions became more violent, her limbs were wrapped in small cushions, and then a strait-jacket put on. The phlegmonous inflammation of the upper limb seemed to be proceeding towards a favourable termination ; the suppuration was less; the walls of the suppurating cavity had a tendency to adhere together; the fever had ceased, and the convulsive movements continued to improve, when fever and diarrhœa returned with greater severity. Phlegmonous inflammation attacked the lower limb also, and within two days it affected the right leg and thigh, and this time it resisted all treatment. Unhealthy pus was secreted, the skin became loose over an extensive surface, the wound ulcerated, the soft parts were destroyed, and the tendons exposed. Numbers of bullæ, filled with a cloudy and purulent serosity, developed themselves on the neck, trunk, and limbs, especially on the arms—some of the diameter of a lentil, and others of a larger size ; ulcers formed on the lips and tongue and in the pharynx even. The fever

became more violent, typhoid symptoms set in, and the patient died about three weeks after the manifestation of the first phlegmonous inflammation.

In this case, as the practitioner who attended it remarked, death was the consequence of the nervous exhaustion, brought on by excessive jactitation and exaggerated by sleeplessness; for the poor child had scarcely four hours' sleep, and even then of interrupted sleep, in the course of the twenty-four hours. The exhaustion was increased by the malnutrition of the patient, who could not feed herself, and, lastly, by the abundant suppuration of the phlegmonous erysipelas from which she suffered, and the starting-point of which, like that of the bullæ, was the adynamia which was consequent on this nervous exhaustion.

Death comes on, sometimes, as a result of cardiac rheumatic complications, as in the following case, which was in one of my wards :—

A young woman, 24 years of age, was admitted into St. Bernard Ward on February 3, 1861. She told us, and her mother confirmed her statement, that on January 1, she had had, with her sister-in-law and her husband, a rather sharp quarrel, which had excited her considerably; soon after this, it was noticed that she was more irritable than usual. On January 15 she had not perfect command over the movements of her right hand, and had some difficulty in sewing and ironing. To this disorder of motility, which rapidly increased in the right arm, there was superadded a certain degree of agitation when she walked. She still continued, however, to attend to her household, and to nurse her last child, who was five months old. In the last days of January the movements of the right side of the body were more disorderly, and became notably more so every day. On her admission her right hand and arm were most affected, and were constantly moved in jerks; her gait was unsteady, and she rested instinctively against the wall or against her bed when she stood up. Sensibility was found normal wherever it was tested, and the patient's mind was clear; the choreic movements of the muscles of her face, and especially of her lips, imparted to her physiognomy a rather strange look. Her manner of speaking was markedly hurried, and her thoughts, although very clear, were extremely versatile. Thus, when it was proposed to take away her child, who ran the risk of being dropped from her arms, she began to cry; then comforted herself, asking that her friends should have the child, and a moment after requesting that he should be left with her. For several months past she had only slept or even dozed for four or five hours every night, complaining of numbness in the limbs, which disappeared only when she shifted herself, or when she got up and walked about. She had never

had rheumatic pain, and no blowing murmur was heard over the heart. Her previous health had been good until January 1. Her muscular strength, when tested with the dynamometer, gave 12 lbs. for the right hand, and 9 lbs. for the left.

For the first two days after her admission into the Hôtel-Dieu, she took two spoonfuls of the syrupus strychniæ. As her agitation persisted, I then gave her syrup of opium, repeated every hour, in order to procure sleep; and although the dose of extract of opium amounted on the first day to 11 grains, she only had four hours' uninterrupted sleep. On the following days, the quantity of opium was gradually increased, but without benefit. The agitation was still extreme on February 9; the patient kept shrieking, and rolling through the ward in search of fresh air; her mind was not affected, for she gave clear answers to questions put to her. But her agitation, the curt and jerked manner in which she spoke, her singular aspect, the constant movements of the muscles of her face, and continued want of sleep for three days, indicated great cerebral excitability. The quantity of opium was again increased on February 9; laudanum was added to the syrup, so that from 9 A.M. to 6 P.M. she took every hour 2 grains of extract of opium; this dose was slightly diminished in the evening. She fell asleep at 12 P.M., after having thus taken from 15 to 17 grains of opium. The next morning her beathing was calm, her pulse was very regular, and had a certain degree of strength (120 to 130); her pupils were contracted, and she was in a deep sleep, from which I did not attempt to rouse her. About half-past twelve, however, her respiration suddenly became embarrassed, and there was some tracheal rhonchus; the breathing suddenly became inappreciable, and the patient who looked as if asleep, died without agony and without fresh convulsions.

A post-mortem examination was made on Tuesday, February 21, forty-four hours after death. There was no notable change in the brain and spinal cord. There was merely slight injection of the cerebral meninges, without a large amount of serosity in the ventricles. The cortical and central white and grey matter were of normal colour and consistency. The pia-mater could be stripped off without lacerating the cerebral substance, and there were no opaline spots in the interlobar fissures. The lungs presented only a few cicatrices at the apex, and were not engorged. The heart was in its normal position, and of normal size and colour. The right chamber of the organ and the pulmonary artery contained no fibrinous clots nor blood-concretions, and the orifices were free and healthy. The endocardium was of pinker hue than normal in the right and left chambers. The aortic orifice was free, and the sigmoid valves healthy. The mitral orifice was of normal dimensions, but the mitral valve

was covered, on its free edge and on its auricular surface, with small polypoid concretions, of pink and yellowish colour, agglomerated, mulberry-like, very adherent, semi-transparent, and resisting pressure. Under the microscope (600 diam. Nachet) they were seen to consist of amorphous granulations and rudimentary fibrillæ of connective tissue. There was valvular endocarditis, and yet there had been no blowing murmur during life, for we had only heard a dry valvular click. The abdominal organs presented nothing worth noting. There were numerous ecchymoses on the arms and legs, and incipient sloughing over the sacrum.

As in the case of other neuroses, *pathological anatomy* teaches us scarcely anything as to the material alterations of the nervous centres in St. Vitus's dance. If you consult various authors, you will find contradictory facts and opinions. One looks upon inflammation or induration of the tubercula quadrigemina as the characteristic lesion of the disease; another regards as such induration or hypertrophy of the brain or of the spinal cord, or a more or less extensive softening of the cerebro-spinal centres; a third believes in calcareous concretions of the brain, a fourth in cysts of the pineal gland, or osteoids of the vertebral canal, and I know not what else. But does not this very diversity of the lesions found after death prove that there is no relation between them and the dynamic phenomena, even if it had not been ascertained that in most cases no appreciable anatomical change can be detected in the nerve-centres? For my own part, in the rare instances in which I have examined the bodies of individuals who had died of St. Vitus's dance, after presenting the most violent symptoms of the disease, I never met with any lesion, I do not mean which could account for death (for in all diseases whatever, in which there is an evident relation between certain symptoms and certain organic lesions, the latter are far from always accounting for the cessation of life, especially in cerebral affections), but which seemed to me to be in accordance with the convulsive phenomena of chorea.

Because tubercles were found in the brain in some instances, no one can infer that this pathological condition is a characteristic lesion of St. Vitus's dance, and even in such cases it may be questioned whether there was any correlation between the tuberculization of the brain and the chorea. I do not, of course, allude to cases in which there were merely choreiform symptoms, for such are no more instances of chorea than epileptiform seizures are of true epilepsy, and the symptoms are evidently dependent, more or less directly, on the appreciable organic alteration. In cases of genuine chorea, the question arises, whether there was not merely a coincidence between this neurosis and the organic lesion in the brain, and whether they

were not both manifestations of a diathesis and nothing more? This view of the question is very plausible, or admits at the very least of discussion, when it is remembered that St. Vitus's dance may show itself in phthisical individuals, in whose nervous centres no tubercular deposit is found after death, although such deposit may be seen in other parts—in the peritoneum, for instance, as in a case of Dr. Rufz, or in the lungs, as in a patient under my care at the Necker Hospital. It is not, therefore, this or that lesion which caused the development of the convulsive affection; but it is the diathesis itself, which not only revealed itself during life by special symptoms, and after death by peculiar anatomical characters, but which expressed itself also by the production of St. Vitus's dance, as it does in other cases by the development of other neuroses.

As to the *rheumatic organic lesions* of the heart and of serous membranes, they are a material proof of the relations which exist between rheumatism and St. Vitus's dance, but they have never been regarded as characterizing the disease.

I wish now to call your attention to the *influence which Intercurrent Febrile Diseases possess on St. Vitus's dance*, and, *vice versâ*, the latter on the former. Dr. G. Sée is the one who has studied this point the most. "While chorea but slightly modifies intercurrent diseases, the latter, febrile affections in particular, have unquestionably an influence on the course of nervous phenomena in general, which has been clearly indicated in the works of the ancients. 'It is better,' says Hippocrates, 'that fever should set in subsequently to spasms than spasms after fever.' In another passage he speaks in clearer terms, saying 'that spasms may be arrested by acute fever,'—an axiom which is fertile in applications, but which has been rejected by many, because it implies important restrictions, which have not been taken into account, and have therefore raised doubts as to the truth of the statement. For if there be instances of chorea on record which was arrested by an exanthematous fever, and afterwards recurred for a time, only disappearing at last rapidly with or without treatment, thus conclusively showing the influence of the fever on the course of the chorea, there are other cases also which clearly indicate that the axiom of Hippocrates may be completely at fault. Thus Dr. Rufz rejects it, and relates two cases of chorea complicated with measles, one of which continued until death without becoming modified.

"The only way to interpret these difficulties, and to conciliate opinions that are so opposed to each other, is to appeal to clinical observation, and submit the facts to a rigorous analysis. Now, of 128 cases which we collected, and in 70 of which febrile complications existed, rheumatic fever was present 25 times, and exanthematous fevers 17 times—namely, scarlatina, 10 times;

measles, 3 times; idiopathic, ephemeral, or catarrhal fevers, 12 times; and inflammations, 16 times (pneumonia 7, angina 3, phlegmonous inflammation 4 times, and diphtheria twice).

"These various diseases, which have but one symptom, fever, in common, exert a similar influence on the nervous phenomena. When these are on the point of disappearing, they are suddenly arrested by the fever, but this is exceptional only. When they are not declining, the fever first produces a general excitation, attended with evident exasperation of the choreic movements, which latter continue as long as the premonitory and invasion stages, and the period of increase of the disease last (from twenty-four to thirty-six hours in the case of ephemeral fevers, and from two to seven days in continued fevers and in inflammations); then, as soon as the fever has reached its point of maximum intensity, the choreic jactitation begins to calm down; and from the time when the reaction ceases, although the pulse is more frequent, and the heat of the skin still greater than in health, the spasmodic movements diminish, and lastly disappear for good—yielding to the efforts of nature alone, and the more easily that the neurosis has been of longer duration. Lastly, in a case of chorea which has just set in, or which is on the increase, the only favourable change is that which takes place in the interval of time which elapses from the invasion of the fever: hence, if the fever lasts for a short period only, and does not allow time for the improvement of the nervous symptoms, the latter persist until the patient's strength is exhausted; and when his general condition is such that his life is endangered, the gesticulations recur until death. In nine cases which ended fatally, the muscular agitation continued in this manner until death, running fatally, as it were, a parallel course to the phases of the intercurrent disease. All these circumstances seem formally to contradict the principle enunciated by Hippocrates; for although it expresses a real and certain truth, the statement is only accurate if the precise moment when the crisis takes place be taken into account. The disappearance of the nervous phenomena does not occur at the outset of the fever, but *generally* after the remission of the febrile symptoms, and on the express condition that the nervous state be on the decline; so that whenever fever is lighted up in a patient who has been suffering from chorea for five or six weeks previously, the convulsive movements will cease: *spasmos febris accedens solvit.* Most of these remarks are applicable to the various kinds of chorea."

I told you, gentlemen, that after lasting a variable time, St. Vitus's dance in most cases got well; the improvement is nearly uniform in its course, the convulsions disappearing in the lower limbs before they do so in the upper extremities. Their

violence goes on decreasing, and there comes a time when they only manifest themselves when the movement which is performed requires a certain degree of energy or a good deal of precision. The face, however, still retains for some time a grinning expression, and the intellect remains weak. At last all these symptoms disappear, and the patient recovers his normal condition.

It is not uncommon, however, that the cure is temporary only; after a variable period of time, a few weeks perhaps, the agitation returns, and there is a *relapse*. In other cases, several months, one, two, or three years, elapse before a *recurrence* of the disease takes place.

It is worthy of notice, that the duration of the complaint in relapses and recurrences is generally shorter than in the first attack. This law of decrease is far from being absolute, however, for the reverse obtains in some cases. Thus Dr. Moynier saw a child, 10 years old, whose first attack of chorea lasted two months, while a second attack lasted two months and a half, and a third and last three months. In another case the first attack lasted two months, the second three, and the third five months. But as the law of decrease applies to the generality of cases, you should be aware of it, and you should take it into account in order to appreciate the value of the treatment which has been had recourse to. From not paying sufficient attention to the natural course of the disease, and from not taking into consideration that after having gone through its different stages, and lasted a determinate period of time, St. Vitus's dance generally got well spontaneously, cures which were entirely due to nature have been ascribed either to methods of treatment based on more or less erroneous theories, or to empirical remedies. Although this is the case in a great many and perhaps in most cases, in some, however, medical interference may be of use, by diminishing the violence of the symptoms, and shortening a little (sometimes very markedly) their duration. It may be especially of use against certain complications, which, if left to themselves, may lead to the most fatal consequences.

Now, gentlemen, what are the therapeutical measures of which we can dispose in the *treatment of St. Vitus's dance*?

I will spare you the tedious enumeration of a great many remedies which have been recommended, based on certain theoretical views which are perfectly erroneous; nor will I say anything of those pretended specifics which have been invented by superstition, or by coarse empiricism, and which are nowadays justly forgotten. I will only speak of those methods of treatment the efficacy of which is recognised, which slightly disturb the natural phenomena of the disease, and make the

patient run the least amount of risks, and which have been adopted by the generality of good practitioners.

The *water-cure*, vaunted first by Dumangin, formerly physician to the Charité Hospital, by Bayle, and afterwards by Jadelot, of the Children's Hospital, consists in the administration of *baths* or the use of *cold lotions*, with water of 10° or 15° Cent. The baths or the lotions are repeated two or three times a day, for one or two minutes each time; and the child is quickly wiped and dressed, and should immediately afterwards take as much exercise as possible. This treatment acts both through the sedative and tonic properties of cold, and through the momentary perturbation of the nervous system which it occasions. It moderates the intensity of the disease, even though it does not arrest it, or sensibly shorten its duration; and from its favourable influence on the whole system, it places the patient in a good condition for going through the attack.

River and *sea-bathing* are other forms of the same method of treatment, and I recollect seeing, at an establishment of mineral baths, an arrangement intended to imitate what is known under the name of *wave-bathing*. The patient was placed on a kind of swing, so arranged that when it oscillated he went very rapidly through the most superficial layer of the water in the tank over which he was balanced.

Cold baths have, however, unquestionable disadvantages. Children, on the one hand, take them with a certain reluctance, and on the other hand, even when they are administered with the greatest precautions, they may bring on rheumatism, which was only threatening, or intensify it if it be already present; in the latter case, therefore, they should be abstained from.

On this account, cold baths were replaced at the Children's Hospital by baths at from 15° to 18° Cent. (59° to 65° Fahr.), and I have myself advised that the child should be merely dipped two or three times into water at first of 24° Cent. (about 75° Fahr.), but the temperature of which was to be gradually lowered every day.

Baudelocque was the first to propose *sulphur-baths*, and to lay down rules for their administration and their indications; their efficacy is sufficiently marked to make most trustworthy practitioners (my colleague, Dr. Blache, among others) adopt them as their chief remedial measure. They should be prepared with from half an ounce to an ounce of sulphuret of potassium dissolved in 100 litres of water, at a temperature of from 30° to 31° Cent. (about 86° Fahr.), and should be taken for an hour at the most. It is essential that they should be repeated with great regularity every day. In cases of threatening rheumatism these baths are contra-indicated.

Besides, gentlemen, the great medical law, on which I every day insist so much (for it finds its application every moment)—namely, the influence of medical constitutions on the results of treatment—also applies to chorea. Thus, Baudelocque and his colleague, M. Bouneau, found themselves compelled, in a period of from eight to ten years, to vary their treatment of chorea: at first the disease was quickly cured by cold water, but a few years later sulphur-baths had to be administered; while these latter again proved of no service after a time, and had to be replaced by preparations of iron.

Among the various methods of treatment of chorea, *gymnastic exercise* certainly holds a pretty important rank; and Dr. Blache has of late made an interesting communication on this subject to the Academy of Medicine,[1] in which he has given the results of his long experience. The idea is not novel, although it has been recently brought forward again; for Dr. Louvet-Lamarre (of St. Germain-en-Laye) published a case[2], in 1827, tending to show the utility of gymnastic exercises. The kind of exercise which he particularly recommended was that of skipping with a rope.

I have many a time heard Récamier speak in terms of praise of the good results which he had obtained from what he called *prescribed and regulated gymnastic exercise*, and which consisted in performing movements in measured time. He thus told choreic children to follow drummers when beating to quarters, and recommended their friends to make them beat time several times in the day. I have often availed myself of this idea of Récamier, and have advised choreic individuals to execute rhythmical movements, guiding themselves by a metronome, or by the pendulum of one of those village-clocks called cuckoos, keeping time to their oscillation. In the beginning partial movements are executed as directed, then combined movements, at first quickly (for they are more easily performed thus), and then more slowly. I have by this means succeeded in modifying not the symptoms of St. Vitus's dance alone, but of other kinds of chorea also, and in particular of the forms of tic which I shall speak of presently.

It would seem as if, in this method of treatment, a strange will replaced, after a time, the patient's will, which was unable to coordinate the movements which itself commanded.

The principle according to which the gymnasiarch deals with the individuals who are entrusted to his care, is exactly similar to

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[1] "Mémoires de l'Académie de Médecine" (Paris, 1855), t. xix. p. 598. See also a learned report by M. Bouvier ("Bulletin de l'Académie de Médecine," t. xx. p. 882).

[2] "Nouvelle bibliothèque médicale," t. xvii. p. 408.

the one which I have just described. He makes them go through certain movements, which he first performs himself before them; and in order to ensure their being done harmoniously, he makes them repeat with him cadenced songs. He begins with simple movements, such as the acts of stretching out and bending the arms, flexing and extending the knees, and striking the ground with the foot in cadence; and when the children succeed in executing these movements with regularity, he tries to make them walk in step, slowly or quickly, and next he makes them run. Lastly, he makes them swing or raise themselves by their arms, going by that means through manœuvres which are gradually more complicated. These exercises are repeated every day, and are not kept up for more than half an hour, so as to be within fatigue. There are certainly great difficulties to overcome in the beginning, but in a short time, and from the first attempts, a certain regularity of the movements is obtained for a few moments, and this improvement becomes more and more marked.

But regulated gymnastic exercise cannot always be managed, and may then be replaced by movements regulated by means of a metronome or of a pendulum, by exercises such as dancing, skipping with a rope, &c., although the latter are not followed by the same beneficial results. It is especially towards the close of the disease that such results are obtained, so that gymnastic exercises are only accessory in the treatment of St. Vitus's dance, and I have more faith, therefore, in the internal administration of remedies.

Of these remedies, some act on the general condition of the system, which complicate the chorea, and influence it more or less. First among these are *tonics* and *preparations of iron*, when the disease is due to chlorosis, which not only accompanies but often precedes it.

On the same ground, again, *arsenic* has been prescribed, from its possessing, as you are aware, the property of causing general excitation, and especially increased vigour of the lower extremities. Dr. Rayer, who has given it in cases of old and obstinate chorea, which had resisted the usual methods of treatment, has thus been able to improve and even to cure them completely.

Yet, gentlemen, although other instances in which this treatment was successfully employed have been recorded by Thomas Martin (who first used arsenic), by Gregory, by Latter, and more recently by Babington, by Hughes, and by Begbie, this drug has been laid aside by these very men who were the first to advocate it, either on account of its difficult administration, and the prudence required, or because the success attending it was really questionable. Yet, let me add at once, arsenic is

administered with greater facility than iodine, and especially than strychnine, of which I shall presently speak.

Iodine and *iodide of potassium* have been likewise vaunted, and are indicated when the object is to modify a strumous diathesis and a predominating lympathic temperament.

Other modes of treatment act directly on the nervous system. One of these is preeminently a sedative plan, which is said to have been formerly used with benefit by Rasori, and without doubt by Laennec, in 1822, and which has been revived within the last few years, after having been forgotten for a long time: I mean the treatment by *tartar-emetic in large doses.*

My learned confrère M. Bouley, in 1857, adopted this treatment with some modifications; and about the same time my regretted colleague, Dr. Gillette, tried it at the Children's Hospital. The results of his trials were published in the following year (1858) by Dr. E. Bonfils, in an excellent thesis, which I advise you to read. After the modifications of this plan which Gillette suggested, and the good results which he obtained, it may be said that the use of tartar-emetic in large doses became of very great importance in the treatment of chorea.

Gillette advised that it should be administered according to the following rules, which Dr. Henri Roger followed rigorously, in the cases which he communicated to the Medical Society of Hospitals, and which were published in the "Union Médicale" for June and July 1858.

The whole treatment generally comprises several series, each of which is of three days, and is separated from the next by an interval of from three to five days. On the first day, tartar-emetic is given in doses of from 4 to 5 grains in the twenty-four hours. This quantity is doubled on the second and trebled on the third day; after this the patient is allowed to rest for three or five days.

If a second series be necessary, that is, if the chorea persist to the same degree, or if the convulsive movements have merely diminished in violence, tartar-emetic is again administered for another period of three days, beginning with the same dose as on the first day of the first series, plus an additional grain.

If, after another interval of rest of four or five days, the disease is not cured, or only incompletely so, the medicine is given for a third time, according to the same rules; that is, the dose given on the first day of the third series will be the same as that administered on the first day of the second series, plus an additional grain. So that if the dose given on the first day of the first series be 4 grains, that on the first day of the second series will be 5 grains, and on the first day of the

third series 6 grains, and on the third day of the third series 18 grains.

I had recourse to this plan, in the case of a patient under my care, in whom St. Vitus's dance was complicated with hysteria, but no improvement was obtained until after several weeks. Dr. Bonfils, who superintended the treatment, did not look upon the case as a successful one, but it is impossible to draw any conclusion from a single instance. A great many cases, however, have been published by Dr. Bonfils, which are not all of equal value, no doubt, but which yet seem to me worthy of drawing attention to the administration of tartar-emetic, according to Gillette's method.

It pretty frequently happens, according to the authors whom I have mentioned, that the chorea improves very markedly after a first series, and in some instances even, if the disease be only of medium intensity, an immediate cure is obtained. But they themselves acknowledge that two or three series in succession are required for a thorough and final cure. Now, if it be borne in mind that the successive series comprise a period of twenty-one days, that the duration of the disease from its commencement is also to be taken into account, as well as the possibility of recurrences, doubts will arise as to the efficacy of the remedy. By carefully reading and analysing the cases published by Dr. Bonfils, it will be seen that the treatment was continued for a period of from fourteen to twenty-five days; that the first manifestation of the disease, when this point was noted, dated two and even three weeks back; lastly, that many of the cases were instances of recurrence of the complaint, which always lasts much less than in previous attacks. It might well be asked, then, what advantages tartar-emetic has over cold affusions, sulphur-baths, and strychnine (of which I shall presently speak), by which the disease may in general be cured; and why, therefore, a plan of treatment should be revived which has been already tried, and then laid aside, and which is somewhat violent in its mode of action, especially in delicate individuals, as many choreic girls are?

Surely, gentlemen, I am, less than anyone else, disposed to doubt the efficacy of the various remedies which are habitually used against chorea, and I admit that the treatment by tartar-emetic is in many cases contra-indicated. But I must also remind you that although chorea yields, in general, to ordinary treatment, and still more to the influence of time, there are yet certain cases, unfortunately, in which the convulsive agitation is so great, that all known remedies are of no avail; and the physician sees unfortunate girls die a miserable death, with an excoriated and deeply ulcerated skin, the result of friction

which no amount of restraint can prevent. Now, should tartar-emetic in large doses be of use in such cases, after all other remedies have failed—(and a certain number of cases tend already to raise the hope that this powerful drug, both perturbing and sedative at the same time, is capable of mastering and in some sort crushing chorea which has resisted all treatment) —even if it should be exclusively restricted to such exceptional instances, therapeutics will be really indebted to Gillette for promising it another chance of success where it was formerly compelled to acknowledge its impotence. The treatment, however, which has seemed most beneficial to me, and which I generally adopt, is that by *strychnine*.

Lejeune had recommended nux-vomica, and Niemann and Cazenave (of Bordeaux) had also, as a last resource, treated by it a case of chorea with complete success, when, in 1831, I myself administered it to a patient suffering from paralysis and chorea at the same time, less with the view of curing his chorea than with that of treating his paralysis.

It was in 1841 only that I laid down distinct rules for treating chorea by this method, and carried on my experiments openly at the hospital. About the same time (without any of us being aware of what the others were doing) Dr. Fouilloux and Dr. Rougier (of Lyons) recommended the methodized administration of strychnine in St. Vitus's dance. While I was taking notes of and publishing cases of chorea cured by nux-vomica, Dr. Rougier published also the results of his researches; but instead of nux-vomica, he recommended the use of strychnine.

Since that time I have myself adopted strychnine, and the preparation which to me seems the most easily managed is the syrup of sulphate of strychnine (one grain of the salt to two ounces and a half of syrup); and I prefer the sulphate to strychnine itself, because the latter is very slightly soluble, whereas the former dissolves to any extent. Two ounces and a half of the syrup are equivalent to twenty teaspoonfuls, each of which therefore contains one-twentieth of a grain of the salt. Two teaspoonfuls are equal to a dessertspoonful, which therefore contains one-tenth of a grain of the salt; and a tablespoonful will contain one-fifth of a grain of the sulphate of strychnine. You must remember that this syrup is not officinal, and you must therefore be careful when you prescribe it. In spite of its bitterness, children do not show very great reluctance to take it.

I now wish to direct your attention particularly to *the mode of administering it*. According to the age of the patient, give on the first day from two to three teaspoonfuls of the syrup, and see that they are taken at equal intervals of time during the day (morning, noon, and evening), so that you may watch the

effect produced, in order not to go beyond a certain point. If the dose of three teaspoonfuls be well borne, it is continued for two days, and then increased by one teaspoonful; after another two days, the dose is again increased by another spoonful, and so on, until six teaspoonfuls are taken in the course of the day—always at equal intervals of time.

When this dose has been reached, a dessertspoonful is substituted for one of the teaspoonfuls; and by attending to the same rules as before, as many as six dessertspoonfuls are administered, containing three-fifths of a grain of sulphate of strychnine. A tablespoonful is then substituted for one of the dessertspoonfuls, and by gradually increasing the dose, with the same prudence, and taking the essential precaution of giving the medicine at perfectly equal intervals of time in the course of the day, you may in the end administer to the child from three-fifths to four-fifths, and even one grain and one-fifth of sulphate of strychnine.

In the case of adults the dose should be larger from the beginning—a dessertspoonful, for example; and it may be gradually increased to as much as two grains of the active principle. But bear well in mind this most important fact, gentlemen—that you should always begin with small doses, and watch their effects, and before increasing should continue them for a couple of days. The treatment should be carefully watched, because the drug must be given in sufficient doses to bring out its physiological effects; and the patient's friends, or the persons about him, should be forewarned of what is to happen.

After a very few days have elapsed, and as soon as the first doses are increased, the patient complains, at certain periods of the day, twenty minutes or half an hour after taking the medicine, of some stiffness of the jaws, of headache, of impairment of sight, of a little giddiness, and of slight rigidity of the muscles of the neck. He complains also that the hairy parts of his person and his scalp itch; the sensation next extends to the non-hairy parts, and in some cases an eruption of prurigo comes out. As the doses are increased the stiffness becomes general, and is most marked in the limbs that are the most convulsed (and these are also the most paralysed, as you know). Muscular jerks occur occasionally also at the same time, and oftentimes spasms and convulsions in hysterical persons. These starts happen in particular when the patient is taken by surprise, or when an order is given him before he has time to will, and they may be so violent that he is thrown down. I remember a young girl, 18 years old, who was under treatment for St. Vitus's dance, at the Necker Hospital, and who, on being unexpectedly addressed by one of the sisters, was seized with tetanic contractions of this kind,

and thrown forwards as by a spring. These tetanic contractions are painful, especially when the patient tries to resist them, and to remain standing; they are instantly quieted, however, on the patient assuming an horizontal position.

When these physiological effects show themselves the doses should not be increased, because strychnine, like all preparations of nux-vomica, belongs to that class of remedies which, by virtue of a special therapeutic influence, and a very remarkable cumulative action, as it were, are apt to give rise to perfectly unforeseen accidents, even though the moderate doses in which they were administered had until then given rise to scarcely appreciable effects.

If it be important that the physician should not be alarmed by the physiological phenomena which he must try to produce, and which, however uncomfortable they may be, are serious only when they are pushed too far (and this never happens if the syrup be properly administered), it is equally important that he should bear in mind that this drug is variously tolerated by different individuals, and by the same individual at different times; so that, even by continuing the same doses, one cannot predict from the effects obtained on the previous day those which will be produced on the next. Thus, six spoonfuls of the syrup may not cause any appreciable physiological effect one day, while, on the next, violent spasms may come on immediately after the first spoonful, even when the same preparation is used, of known strength. I need not add, that when the administration of the first spoonful brings on spasms, the medicine should be stopped for the day. There being nothing to account for such results, I tried to make out whether meteorological conditions had any share in their production, but my inquiries led to no conclusions.

This variability in the degree of power of the drug renders its administration a delicate matter, and demands the most scrupulous care; and on this account perhaps this method of treatment will not obtain the importance which its unquestionable advantages ought to give it. The reluctance with which it is had recourse to is all the greater from the fact that it should be persisted in for several days after the chorea has ceased, in order that its influence be complete. By beginning it again in smaller doses, and for a shorter period, after an apparent cure, relapses may be prevented. This is a rule which I have laid down for myself, but which it is impossible, or very difficult, at least, to follow in hospitals. I shall merely say a word on the use of *electricity* in the treatment of St. Vitus's dance. De Haën was the first to recommend it, and his method consisted in drawing sparks from the spine by means of an electric machine or of a Leyden jar. This mode of applying

electricity is nowadays justly abandoned, nor has *electro-puncture* been more successful.

As to the good results which are said to be obtained from *faradization of the skin*, I have never been able to verify the accuracy of the statement, and I have not been convinced of its utility by the perusal of cases in which it had been used. I hesitate before having recourse to it when I find that the treatment, in five out of eight cases, lasted from twenty-four to forty-seven days; and when, on the other hand, I hear from the very advocates of the plan that it is attended with certain disadvantages; that it causes such pain, for instance, that several patients had to be rendered insensible by chloroform in order to be faradized.

As you may imagine, *antispasmodics* and *narcotics* have been used against chorea, such as *valerian*, *camphor*, *assafœtida*, *musk*, &c., which have been alternately recommended, put aside, and tried again. Of late an interesting memoir has been published by Dr. Corrigan, in the London *Medical Times*, on the use of *Cannabis indica*. His first case is that of a little girl, 10 years of age, who had been ill for five weeks. She took five minims of the tincture three times a day, and in eleven days a considerable improvement followed; the dose was then gradually increased to twenty-five minims three times a day, and the patient was discharged cured in a little less than five weeks. The subject of the second case had been ill a month, and had to be kept under treatment for forty days; she also took twenty-five drops of the tincture three times a day. Lastly, a young girl, aged 16, who had been choreic for the previous ten years, was cured in a month.

These cases are not very conclusive, as you see; but I will again say what I told you regarding tartar-emetic. *Cannabis indica* unquestionably possesses an alterative action on the nervous system, and may therefore prove an additional resource in cases of obstinate chorea, and whenever narcotics are indicated with the view of preventing certain dangerous complications.

I have already told you, gentlemen, that death may be the result of extreme agitation, aggravated by sleeplessness, in St. Vitus's dance. Now, chloroform inhalations have been used with benefit by M. Fuster against this agitation.

When there is obstinate want of sleep, which gradually exhausts the patient's strength, I have recourse to *opium*, which I gave, as you saw, to the patient in bed 20, St. Bernard Ward. I administer it in large doses; and this patient took, for several days in succession, a tablespoonful of syrupus opii [1] every four hours.

[1] This preparation contains one grain of ext. opii in an ounce of syrup.—Ed.]

In more severe cases I prescribe still larger doses of opium.

On September 20, 1842, a young woman, aged 20, was admitted into the Necker Hospital (bed No. 27, St. Anne's Ward). She was pregnant, and was suffering from a first attack of chorea, which had set in for the last eight days. Her convulsive agitation was extreme: her limbs, trunk, and eyes were continually moving. Her right leg and arm were paralysed; her ideas were somewhat confused, and she was strangely talkative, a circumstance which was all the more remarkable that her tongue was affected, and her articulation embarrassed. The pupils were moderately dilated, but sight was good on both sides. Besides having no appetite, the patient could not feed herself; and she could scarcely chew and swallow her food when she was fed. There was no disturbance of the digestive organs except constipation. On the day of her admission I gave her two grains of alcoholic extract of nux-vomica, and six grains on the following day. The physiological effect of the drug showed itself five hours after the administration of the first pill, and lasted an hour and a half. A second pill was, however, given, notwithstanding this, three hours afterwards; but before an hour and a half had elapsed, tetanic jerks supervened, during which she screamed out, and the attack lasted from half- past 7 to 12 P.M. The jerks, in the intervals of which the choreic convulsions returned with still greater violence than before, were such, that the patient jumped up in her bed, and her respiration was interrupted at each paroxysm, her face becoming at first pale and then livid. A strait-jacket had to be used in order to restrain her, and she had it on when I saw her the next morning.

On seeing that, instead of being quieted, the patient's agitation had been so exaggerated that she had not torn, but worn out, through the violence of her movements, her chemise and the bed-clothes, and had excoriated her back, I stopped the nux-vomica, and, on account of her want of sleep and her extreme exhaustion, I prescribed for her a mixture containing four grains of *sulphate of morphia*, a fourth part of which was to be taken for a dose, and the whole in twenty-four hours. The patient took three doses, and an hour after the first she fell asleep quietly, and slept for two hours. When she awoke, she remained pretty quiet for four hours; but on her getting excited for some cause or another, she was again as violently convulsed as before, so that the remainder of the mixture was given her during the night, and she slept till six in the morning.

The choreic symptoms then returning again, I doubled the quantity of morphia, making it eight grains. But it was remarkable that the improvement of the previous day was less easily

produced this time. The agitation was greater than ever, and although the patient dozed a little after taking the whole of the mixture, she was so excessively agitated in the evening, that my clinical assistant thought fit to prescribe another mixture, containing two grains of sulphate of morphia, and made her take several spoonfuls of it, one after another, in his presence. She became markedly quieter, and fell asleep. Her rest was disturbed at first, but became quiet for the rest of the night after she had taken a few more spoonfuls of the mixture.

The next morning, when she awoke, the convulsions returned with nearly the same violence, and I increased the quantity of morphia to 12 grains. For two days she took this dose; and on the agitation appearing again, I successively increased it to 20, 25, up to 30 grains. This last quantity was even given in two doses, but the first dose alone was kept, the second was vomited. In spite of this, the same quantity was repeated for two days, and the patient bore it well. The disease at last yielded completely; the patient's sleep became calm and natural, the choreic movements were very slightly marked, and the young woman, feeling comparatively well, requested her discharge on October 17, that is to say, after a stay of twenty-seven days in the hospital.

You see, gentlemen, what enormous doses of opium can be given in grave cases of chorea. I gave another woman in the Hôtel-Dieu fifteen grains of sulphate of morphia, but I do not remember ever prescribing such a large dose as the one I gave my patient in the Necker Hospital.

While on this point, let me tell you that medical men dread too much, in my opinion, the use of opium in large doses, in St. Vitus's dance and other grave neuroses; and, indeed, in all cases in which it is indicated. They forget the precept laid down by Sydenham in his letter to Robert Brady, and which he repeats in his admirable letter to William Cole on the subject of smallpox, namely, that 'the dose of a remedy should be increased and repeated in proportion to the intensity of the symptoms' (*remedii dosis et repetendi vices cum symptomatis magnitudine omnino sunt conferendæ*). A dose which may be powerful enough to remove a slight symptom will not have any influence on violent symptoms, and a dose which may endanger the patient's life in certain cases will in others save him from certain death. (*Quæ enim dosis remissiori symptomati coercendo par est ea ab alio fortiore superabitur, et quæ alias ægrum in manifestum vitæ discrimen conjiciet, eumdem ab orci faucibus liberabit.*)

I have often related the case of a brush-manufacturer, who in 1846 consulted me, on account of excessive nocturnal pain in his bones. He had come to take from about six to eight

ounces of Rousseau's laudanum, a preparation which contains three times as much extract of opium as the laudanum of Sydenham.[1] He drank it in tumblers in *my presence*; and added, that on his trying the sulphur baths at Enghien, his pain had been so intensified that he determined on poisoning himself, and took, in one dose, twenty-four ounces of Rousseau's laudanum, that is to say, *more than two ounces and a half of the aqueous extract of opium. He slept for three hours only.*

About twenty years ago, I asked Prof. Andral to see in consultation with me a young man, a friend of mine, who was suffering from an extremely painful neuralgia. We prescribed opium pills of one grain each, which were to be taken until the pain had been subdued. He took twenty-four pills in the space of twelve hours (that is to say, twenty-four grains of gummy extract), and got perfectly well. He was only slightly narcotized; and now that he no longer needs this remedy, he could not, any more than any other man, take even moderate doses of it without feeling some inconvenience from it. You are aware that in cerebro-spinal typhus, Dr. Boudin gives opium in large doses, proportionately to the gravity of the nervous symptoms. He begins with ten and even twenty grains of the gummy extract, which he gives in one dose, and then repeats every half-hour smaller doses of one and two grains, until the patient falls asleep. Such examples show, therefore, that in the administration of opium, the dose of the medicine is to be less taken into account than the effects which it produces. This is what Peyrilhe meant by saying that when a man is as awake as four, he should take as much opium as five, in order to sleep as one.

In grave forms of St. Vitus's dance, therefore, when it is demanded by the excessive agitation and the absence of sleep, opium should be given *largâ manu*. Yet do not believe that this treatment is infallible. It has sometimes failed in my hands, but in such cases, the patients did not only suffer from convulsive agitation in the extreme, and non-febrile delirium, but there was fever present as well as delirium, and nervous symptoms which do not belong to chorea, generally cerebral rheumatism, and opium was powerless against them, as in the sad case which I related to you in the course of this lecture.

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[1] The laudanum of Sydenham is the vinum opii compositum of the French codex. The following is the formula of its preparation:

Opium	ʒ ij.
Saffron	ʒ j.
Cinnamon . . .	ʒ j.
Cloves	āā.
Malaga wine . .	O j.

20 minims are equivalent to about one grain of ext. opii.—Ed.]

Lastly, gentlemen, *hygienic measures* play an important part in the treatment of St. Vitus's dance. Thus, nutritious and tonic food taken at regular intervals, open-air exercise, within fatigue, so as to facilitate the organic movements of repair, and to prevent the recurrence of the disease, cold bathing and swimming, are formally indicated.

In severe cases of chorea, certain precautions should be taken in order to prevent the patient from hurting himself in his disordered movements. The bed on which he lies should be of sufficient width and thickness, and shut in on the sides by padded flaps, so as to save him from falling. In those extreme cases in which the poor child tears and rubs off his skin, by continual friction against the bed-clothes, and when the agitation is such that he is thrown out of bed, over the flaps, a strait-waistcoat is sometimes had recourse to; but instead of diminishing the risks which are dreaded, the chances in their favour are increased, because the strings give rise to excoriations of the skin, which afterwards turn into horrible wounds.

For my part, I allow my patients all freedom of action; but I place them in conditions which prevent their hurting themselves. When I was physician to the Children's Hospital, I invented a sort of apparatus which is still used now. It merely consists of a large box, made of deal or of oak, about 2 metres long, $1\frac{1}{2}$ metres wide, and $1\frac{1}{4}$ metres high, padded with thick and soft cushions on the sides and at the bottom. The child, when placed all naked inside this box, may move about freely without fear of any accident. To protect him against the cold, sheets are either thrown over him, or are made to close the upper part of the box; or a better plan consists in putting hot-water bottles between the walls of the box and the cushions. Those boxes are easily procurable for a small sum, and may thus be used in poor families as well as by the rich.

Another simple means, namely, waddling the child, is of great utility in very grave cases. It is now several years since it has been recommended, but it is, in my opinion, too rarely employed. The upper and lower limbs of the child are first carefully wrapped in wadding, which is maintained by a bandage, and then the lower limbs are kept closely approximated, and the arms fixed along the sides of the trunk by means of bandages again. I need not add that the turns of the roller which are meant to confine the arms, should not be so tight as to interfere with respiration. In general it is found necessary to apply the bandage twice in the course of twenty-four hours. It is a fact that, in the majority of instances, the forced rest in which the muscles are kept calms the extraordinary agitation of some patients. This plan is, of course, had recourse to in very grave forms only.

OF THE DIFFERENT FORMS OF CHOREA.

Chorea saltatoria.—Methodical or Rhythmic Chorea.—Tic-douloureux (*chorea neuralgica*).—Tic non-douloureux.—Writer's Cramp (*chorea scriptorum, functional spasm* of Dr. Duchenne, de Boulogne).

Gentlemen,—A short time ago, one of my most eminent confrères and I differed as to the diagnosis to be made in the case of a patient who had for more than a year been afflicted with choreic movements. My learned colleague called the disease chorea (meaning thereby St. Vitus's dance), while I was of opinion that it was a form of chorea, but not St. Vitus's dance.

Now, I based my opinion on these facts. By questioning the patient's father and the patient himself (a boy, 12 or 13 years of age, and full of intelligence) on the character of the symptoms, I made out that the voluntary movements remained somewhat regular in the midst of these choreic convulsions. Thus the boy declared that he had not lost his usual agility, that he could leap without difficulty, and as well as any of his companions, over barriers; that he could, when going up a staircase, take three or four stairs at a time; that he had no difficulty in skipping with a rope; and lastly, that he used his hands as well as anybody else to feed himself, and even to drink; all which actions cannot be performed, as you know, by persons suffering from St. Vitus's dance.

From some obscure disturbance of his nervous system, this child executed curious movements, and was thrown forwards, as if by a spring, by involuntary muscular contractions, which made him jump to seven or eight feet in front of the place where he might be standing, or get up abruptly, mechanically (if I may use the expression), from the chair on which he might be sitting; he never fell down. There was a kind of harmony amid this disorder of the locomotor functions, for if all the muscles contracted independently of the will, they all acted simultaneously at least. This, therefore, gentlemen, is a form of chorea which differs much from St. Vitus's dance, and to which the name of *chorea saltatoria* has been given.

A few years ago, another instance of the kind came under my notice. A boy was brought to my consulting-room by his father, who had begun to relate to me his case, when he suddenly got up, as if pushed by a spring, jumped on a piece of furniture with marvellous suppleness and agility, and then returned to his chair and sat down quietly. What he had done had shown me the nature of his case, which his father was going to describe less clearly to me. His illness had lasted some time; these singular attacks had set in suddenly, and his

intellect had not suffered in the least yet; in the intervals between the paroxysms he was as quiet as possible. He got perfectly well. Although, as in both the above cases, there rarely is an apparent impairment of the intellectual faculties, *chorea saltatoria* seems to me, however, to belong to the same great class of mental disorders as *tarentism* and the *epidemic dansomania* of the middle ages. It is only a variety perhaps of the *methodical* or *rhythmic* forms of chorea, which include *chorea festinans* or *procursiva*, *chorea rotatoria*, and *chorea vibratoria*.

In *chorea festinans* the individual is irresistibly impelled to run forwards, without being always able to avoid obstacles, or, on the contrary, to go backwards continuously without being able to help himself. This affection should not be confounded with the semi-delirious condition under the influence of which individuals, who are threatened with certain brain attacks, or who are just recovering from an epileptic fit, are carried along in spite of themselves.

In July, 1861, I saw, in consultation with Dr. Duclos, a retired military man, about 60 years of age. He was walking with his brother along the banks of the St. Martin canal, when all of a sudden, without any warning, he began to walk with extreme rapidity, and almost to run. His brother in vain called out to him to moderate his step; he walked quicker and quicker, scarcely avoiding the obstacles in his way, and it was only with difficulty that he could be restrained after more than ten minutes. He stammered, looked strange, and a few moments afterward became slightly hemiplegic in consequence of hæmorrhage into his brain. It is pretty probable that the first impression produced on the brain by the laceration of its substance was the intellectual disorder manifested by his mad running. The most curious case of *chorea festinans* which has come under my observation is that of a Hâvre merchant, who came to consult me in May, 1860. He was with some other persons in my waiting-room, and he got up and trotted into my consulting-room, when his turn came, in such a curious manner that he raised a laugh among the others. His body was stiff and inclined forwards, with his arms hanging straight down along his trunk and thighs, while his eyes were fixed. He ran quickly on tiptoe, taking small steps, as if in fun. When he got near me he stopped and sat down without difficulty. I had seen enough in order to recognise the strange neurosis from which he was suffering. He then told me that these symptoms had come on almost insensibly for about a year; he could no longer go out, felt bodily and mentally weak, and could scarcely conduct the business of his firm. His speech was a little thick. One might, at first sight, think of incipient

general paralysis, but with a little care chorea procursiva could be recognised. After he had told me his story, I made him get up and walk slowly, pressing down his foot. He had some difficulty in starting, and seemed fixed to the ground, but still he took the first step forward by himself, and walked several times round my consulting-room *slowly*. He could therefore command his movements by an effort of the will, while this is not the case in general paralysis or in *tremor senilis*, St. Vitus's dance, or locomotor ataxy. I found by testing it that his cutaneous sensibility was normal, and his muscular power, tried with Burq's dynanometer, showed no diminution, while, as I shall tell you on another occasion, the muscular power in *paralysis agitans* (of which, at the end of the year 1860, you saw so curious a case, that of the woman in bed No. 2, St. Bernard Ward) may be so considerably diminished as to mark only 10 lbs. with Burq's dynamometer.

I prescribed for that gentleman ten turpentine capsules a day (containing about 100 minims), which he was to take for twelve or fifteen days a month, and, in addition, I ordered warm baths of several hours' duration.

Two months later, when I saw him again, he had improved considerably; I then sent him to the Néris baths, and he had so improved on his return, about the month of August, that I might have hoped for a complete cure, if I had not been aware how obstinate this neurosis is. Yet he could go into the streets, attend to his business, work, and write, but had always a certain tendency to trot on starting. He restrained himself at once, and could walk more quietly, although with a look of effort and restraint. On several occasions I made him walk in step like a soldier in my own room—and this is a very difficult kind of walk, which requires great precision of movements. He spent the winter of 1860–61 pretty well, and when I saw him again at the end of May, 1861, he had not lost ground, and I sent him to the Néris baths a second time.

I believe that, in some instances, general paralysis and *paralysis agitans* have been confounded with *chorea festinans*, but I regret that I have not in my possession notes of cases sufficiently distinct and free from complications that I might give you a complete sketch of this affection.

Chorea rotatoria is characterized by rotation or oscillation of the head, or trunk, or of one limb, recurring from 20 to 30, 40, and 80 times a minute. It sometimes terminates in death, and spares neither age nor sex, although it occurs less frequently in children.

Chorea oscillatoria consists in irregular or measured oscillations, partial or general, of the head, trunk, or limbs.

These singular affections must surely recall to your mind,

gentlemen, another kind of partial chorea, which is very common, and which goes by the familiar name of *tic*. I do not mean *tic-douloureux*, *chorea neuralgica*, or *epileptiform neuralgia*, of which I spoke at length in a previous lecture, but *tic non-douloureux* (spasmodic tic), which consists in instantaneous, rapid, involuntary contractions, generally restricted to a small number of muscles, those of the face usually, but which may also affect the muscles of the neck, trunk, or limbs. Everyone must have seen such cases. Thus, there may be only rapid winking, a convulsive pulling of the cheek, of the ala nasi, and of the commissure of the lips, which gives to the face a grinning look; or there may be nodding of the head, abrupt and transient contortion of the neck recurring every minute; or again, the shoulder is shrugged, and the abdominal muscles or the diaphragm is convulsively agitated; in a word, the disease may produce an infinite variety of strange movements which baffle all description.

The complaint is essentially chronic, and is, so to say, part and parcel of the individual's constitution; he is the only one, sometimes, who does not notice it: it is cured with difficulty: but it is a strange circumstance that it may shift from one place to another. When by treatment, and by exercising the affected muscles, a tic has at last been cured, it may soon reappear elsewhere; thus, it may leave the face, for instance, and seize on the arm or leg. I was lately consulted by a young Englishman who had come from Dieppe, and who was suffering from convulsive and violent movements of the head and right shoulder. After submitting for some time to the methodical gymnastic exercises which I prescribed for him, the tic disappeared from the right side, where it had for a long time been located, but shortly afterwards showed itself in the left shoulder. You remember what I mean by prescribed gymnastic exercises, and which consist in executing movements, according to order, with the convulsed muscles, and doing so regularly, keeping time to a metronome or a clock.

In some cases of tic the patient utters a more or less loud cry, which is very characteristic. Once I recognised one of my old schoolfellows (after an interval of twenty years) as he happened to walk behind me, through a sort of barking noise which he used to utter in our school-days.

The tic may consist in this cry or bark alone, which is a true *laryngeal* or *diaphragmatic chorea*; there is, besides, a singular tendency always to repeat the same word or exclamation, and the person even speaks out loudly words which he should like to keep back. This complaint is very often hereditary. I was consulted by a lady from Burgundy who had spasmodic tic of the face, while her three daughters were suffering from tic

affecting muscles in various portions of the body; and the poor mother, who was deeply grieved at the infirmity of her three daughters, and did not notice her own, reproached them with their nervous movements with a bitterness which was curious to see. The hereditary influence may show itself in a different manner. By carefully questioning a patient suffering from tic, you may sometimes find that his ancestors, direct or collateral, were all subject to very different neuroses.

I saw very recently a boy, 14 years of age, who was afflicted with extremely severe tic, throwing his head sideways with an excessively abrupt gyratory motion, and uttering a small sharp cry. I had seen him before during the summer of 1860, and he then used to utter fierce cries every moment, without his mind seeming to be in the least impaired. This sad condition had lasted several months, and had seemed to improve under the influence of atropine alone. His eldest brother had, for several years, suffered from facial spasm characterized by grimaces, during which all the muscles of his face were violently convulsed. His father has been affected with locomotor ataxy for the last twenty years; his paternal grandfather committed suicide in a fit of monomania, and several of his relations, on his mother's side, have been insane.

Writer's cramp or *chorea scriptorum* is the name given to an affection for which Dr. Duchenne (de Boulogne) has proposed that of *functional spasm*.[1] It is sometimes a consequence of the over-use of certain muscles, and comes on when these muscles are called into action either instinctively or voluntarily. Thus, it attacks individuals who write continuously, for a prolonged period, or with excessive rapidity. It sometimes consists in a spasm, a voluntary, continued, and more or less painful contraction of the extensor and flexor muscles of the fingers, and to such cases the term writer's cramp is perfectly applicable; but at other times it is true chorea; when the individual wishes to write, his fingers move more or less violently, shake, or are actually convulsed, so that they are unable to finish what they began to write.

Dr. Duchenne (de Boulogne) states that this affection (which is also attended with paralysis) may not only affect the hand, but any other part of the body also, and it is on this account that he proposes for it the name of functional spasm, a denomination which, however open to criticism, has yet the advantage of not particularizing, as that of writer's cramp does. He relates a certain number of cases showing the different localities in which the complaint may be seated.

"In writers it may extend to the muscles of the forearm, the

[1] "De l'électrisation localisée et de son application à la pathologie et à la thérapeutique." 2e édition. Paris, 1861, p. 928.

hand performing a movement of supination as soon as the patient tries to write a word, so that the pen is turned upwards without his being able to prevent it.

"In the case of a tailor, the arm turned violently inwards, through contraction of the sub-scapularis, as soon as he had done a few stitches. He never had this annoyance when he made any other movement.

"A fencing-master found that as soon as he placed himself in a posture of defence, the arm with the hand of which he held his sword, turned immediately inwards.

"A turner complained that the flexor muscles of his foot upon the leg were thrown into contraction, as soon as he placed his foot on the footboard of his lathe; but he never felt the same thing when he walked or performed other voluntary movements with his leg.

"In the case of a labourer, a paver, both sterno-mastoidei contracted during the instinctive action of the muscles which keep the head in equlibrium in an intermediate condition between flexion and extension. They did so with such violence, that his head bent down with excessive force. He had only to rest his head against anything in order to stop all contraction; and none took place also when he lay down, or reclined backwards, leaning his head against the back of a chair.

"A *savant*, who had spent several years translating manuscripts, complained of the following symptoms which had come on, for the last six months, whenever he read or looked fixedly at anything. His sight, which had been good until then, and which even then was good when he looked about, grew dim whenever he looked at any object for a few seconds. He had double vision, and it could be easily seen that this was due to the spasmodic contraction of the internal rectus of the left eye, which disappeared as soon as he ceased to look fixedly."

The most curious instance of this singular neurosis, which has come under Dr. Duchenne's observation, occurred in a country priest, whose inspiratory muscles were affected. During inspiration, the whole right side of his abdomen was alternately tense and depressed, while his epigastrium swelled out normally on the left side. A medical man had diagnosed paralysis of the right half of the diaphragm, but the paralysis was merely apparent. The disturbance in the breathing was solely due to the spasmodic and painful contraction of the abdominal muscles on the right side, and of the obliquus externus especially, for at each inspiration this latter muscle could be felt to harden, and the direction of its contracted bundles could even be traced through the emaciated integuments. The spasm was so violent, that the body turned from right to left at every inspiration: it

was accompanied with pain, and was a true cramp, which lasted during the whole period of inspiration. This conflict between the inspiratory and expiratory muscles prevented the epigastrium and the base of the chest from expanding on the right side, and consequently prevented the lung from dilating. Hence it was that breathing was considerably impeded, and that the patient had always a choking sensation. There was no fever, and for two years no treatment gave relief. Faradization failed like the rest.

I will quote another case in illustration, and from Dr. Duchenne's work again. A Strasburg student, M. V——, overworked himself when preparing his examination for the degree of *bachelier*. The excessive strain on his mind, and the efforts which he made to resist sleep, gave rise, according to his statement, to a sensation of painful constriction in the temples, forehead, and eyes, so that he had been obliged to discontinue his studies. He could not begin reading without this sensation returning at once. Dr. Duchenne found that at such times the eyebrows were pulled up through the contraction of the frontal muscles, and that the eyelids were closed by the contracting orbicular muscles, while the face flushed, and the temporal veins swelled. This condition lasted several years, and was brought on by reading alone. The young man committed suicide at last, in despair of ever getting well.

Indeed, gentlemen, whatever its seat may be, this complaint is incurable. Absolute rest of the affected muscles can alone prevent it from returning. All treatment has failed. Yet persons suffering from writer's cramp can still write sometimes, by using a peculiar penholder invented by Dr. Cazenave (of Bordeaux), and the description of which has been given[1] by Valleix in his "Guide du Médecin Praticien." I have told you that Dr. Duchenne (de Boulogne) is of opinion that functional spasm may be also characterized by paralysis, and he relates two cases in support of his view, in the memoir which I have quoted. One is that of a book-keeper, whose adductor pollicis lost all power after he had written two or three lines, so that he dropped the pen. He could only write by holding the pen with his index and middle fingers. Yet the muscle could act with energy whenever he had not to hold a pen: there was no muscular spasm in this instance. In the second case, the functional paralysis was seated in the infraspinatus muscle, preventing the arm from rotating from without inwards, and consequently the forearm, when flexed on the arm, from executing the same movement.

[1] Paris, 1860, t. 1, p. 906.

HYSTERICAL CHOREA.—HYSTERICAL COUGH.

Gentlemen,—I alluded, in a previous conference, to the case of a girl 13½ years old, who occupied bed No. 6, in St. Bernard Ward, and who was suffering from hysterical choreiform convulsions. About the same period, you could see another case of the kind, namely, a young girl 18 or 19 years old, who lay at No. 33, in the same ward.

The invasion of the disease, in the latter case, had coincided with a sudden suppression of the menstrual flux, in consequence of a fright. Convulsive agitation had immediately shown itself, together with jerking movements of the limbs and trunk, so violent as to prevent her from standing. Her tongue was similarly affected; hence she was unable to connect the syllables together, although she could articulate them separately. She stammered in a singular manner, repeating with extraordinary volubility, and for a pretty long time without stopping, the last syllables of the words which she attempted to say, articulating the first syllables with difficulty. It was a remarkable fact, however, that she did not stutter when she sang, and no modification of speech could then be suspected. I at first thought that she was feigning; but this idea could be entertained with difficulty, in presence of convulsive phenomena which lasted a whole day, without a moment's interruption, and ceased during sleep only. On reflecting, however, how painful it is for a healthy individual to move a limb for several minutes, and *à fortiori*, to agitate it in the same manner as this young girl did, it could be understood how impossible it must be to act such a part during sixteen or eighteen hours out of the twenty-four, and without interruption.

There was a third patient in bed No. 11, who, from her appearance, looked more like a girl 15 or 17 than 12½ years of age, as she really was. The attack for which she had been admitted dated only two days back; but she had felt the first symptoms of the complaint six months previously. Her mother was subject to convulsive seizures; one of her brothers, 4 years old, had had several similar ones also; and from her description of the fits they must have been epileptic. Her health had been good until six months ago, when she was suddenly seized, without any known cause, with violent pain in the head and very abundant hæmorrhage from the nose, after which she had become extremely weak. Two or three days afterwards her abdomen had swollen considerably, and she had suffered from colic and gastralgia. Her appetite was good, however, her digestion regular, and taking food neither increased nor diminished the pain in the stomach and abdomen, while the

swelling of the latter varied very much. On her admission I found that her abdomen was swollen out to the size of that of a woman in the eighth month of pregnancy; and the tympanitic resonance heard all over it on percussion was proof sufficient that the distension was due to meteorismus. She complained also of pain in the dorsal region, in the loins and the lower extremities, which she spoke of as cramps in the latter regions. Lastly, the headache continued still.

She took very little notice of the above symptoms, when, two days before she applied for admission, she had, without appreciable cause, and without any antecedent emotion, what she termed *a nervous attack*, which still persisted when I saw her. This consisted in convulsive movements, which were at first confined to the arms, and extended to the legs twenty-four hours afterwards. You must have remarked, gentlemen, how, in spite of the choreic convulsions which agitated the limbs during this true chorea, the movements that were performed, however involuntary they might be, were executed with regularity and in harmonious combination. Besides, contrary to what takes place in St. Vitus's dance, they stopped when the patient was asked to stretch out her arm; she could perform the latter movement with the greatest facility, and in a perfectly straight line. She could take hold, with ease, of any object shown her, reached it directly, and never dropped it after getting it in her hand.

Cutaneous sensibility was abolished in certain regions of the body: over the back of the forearm, along the outer aspect of the left thigh, in certain portions of the face and of the chest, there was *analgesia*; when she was pricked with a pin she felt a mere touch, and had not the sensation of pricking. No doubt could exist as to the nature of her complaint, for she had on several occasions regular *hysterical attacks*.

I met in consultation my colleague and friend Dr. Horteloup in the case of a young lady 19 years old. She had received an excellent education, professed sentiments of the purest morality and of the most enlightened piety, free from all ridiculous show of outward devotion, and was, in one word, a person of sense, whose intellectual and moral condition removed all idea of deceit and of those grimaces with which hysterical girls seem so unaccountably anxious to deceive the persons about them, and even their medical attendants when they can. This young lady had lost, eight or ten months previously, a sister to whom she was deeply and tenderly attached. Her grief was all the greater that she keenly felt for her mother as well as for herself. Since that time she had been subject to strange convulsive movements of the head and upper limbs; yet, when she came to Paris to consult Dr. Horteloup,

who had attended her on a former occasion, she was less sad, looked more cheerful, and was pretty easily diverted from her gloomy ideas. When I saw her, her aspect was that of perfect health, but her whole left side was the seat of violent choreic movements—so violent, indeed, that she was in danger of hurting herself against the neighbouring pieces of furniture or the walls. If one attempted to arrest these movements by taking hold of her hand, they grew worse, and were accompanied with a sense of pain, and most unpleasant general *malaise*. There was one means, however, of quieting all this agitation, as if by magic—namely, by asking her to play the piano. She could spend an hour or two at the instrument, playing to perfection, and with the greatest regularity; in excellent time, and without missing a note. She played a piece in my presence with marvellous facility; and this single fact, even in the absence of other proofs, would have sufficed to show me that this kind of chorea had nothing in common with St. Vitus's dance; for no one suffering from this latter disease is able to do what this young lady did. These illustrations, which I might multiply, if it were necessary, suffice to show you the difference which exists between St. Vitus's dance and hysterical chorea. In the latter affection I repeat, however powerless the will may be to prevent the disorderly contractions of the muscles, it can still command combined movements, and cause them to be executed with regularity and harmony. When the patient walks she trots, it is true; but she follows any line which she chooses without deviating from it. If she wishes to carry her hand in any direction, she reaches the end she has in view directly and without difficulty, although her arm may be convulsively agitated; if she tries to seize an object, she does so at once, without erring; and when she has once caught, she never drops it, and can carry or place it wherever she likes. I have told you how different the case is in St. Vitus's dance.

Thus, if we merely look at the form of the choreic phenomena, it is easy, with a little attention, to distinguish these two kinds of chorea one from the other, as their nature is so essentially different.

It very rarely happens, besides, that the former is not accompanied, preceded, or followed by some more special and characteristic symptoms. In the absence of its great manifestations, of its convulsive seizures, hysteria shows itself by that group of perfectly special physical or mental dispositions which some authors term *hystericism*; or there are certain local phenomena proper to the disease, such as that strange sensation of umbilical and epigastric constriction, as if a foreign body were going up from the œsophagus to the throat, producing there a sense of choking, to which the name of globus hystericus has

been applied; or, again, perversions of cutaneous sensibility, which is sometimes exaggerated in certain parts of the body, giving rise to the so-called clavus hystericus, and sometimes, on the contrary, diminished or entirely abolished (analgesia and anæsthesia).

Hysterical cough, which is nothing but a convulsion of the muscles of the larynx and diaphragm, presents great analogies to these forms of chorea. However convulsive it may be, it resembles in nothing other convulsive coughs; for instance, the convulsive cough properly so-called, which is so frequently observed in children, or that of hooping-cough. It is not, like them, attended with those violent spasms which cause fits of choking, threatenings of asphyxia, and give rise to pulmonary or cerebral congestions.

A young woman, who occupied for a few days bed No. 1, in St. Bernard Ward, was subject to this cough; and you had an opportunity of verifying the accuracy of the statement made by my excellent friend, Dr. Lasègue, in his "Memoirs on Hysterical Cough,"[1] how this kind of cough, when uncomplicated, resembles that which is excited by the inhalation of certain gases—chlorine for example. It is sometimes preceded by a sensation of tickling in the larynx, is dry, or with a trifling mucous expectoration, sonorous, and of a somewhat monotonous rhythm. The patient either coughs at every expiration which succeeds an inspiratory movement, or makes two, three, or four coughing expirations before she begins to breathe again. In the intervals between the paroxysms, the breathing is less deep than usual, because the patient dreads deep inspirations, which render the cough more troublesome; but there is no dyspnœa, and, on auscultation, no other modification of the normal respiratory sounds is detected than a slight diminution of the vesicular murmur at the moment when the inspiratory effort is withheld.

While it lasts, an hysterical cough has the same rhythm and *timbre*. The jerks constituting the paroxysm are sometimes so often repeated that it seems as if the latter consisted of a single cough instead of a series of coughs; but there are intervals of rest between each paroxysm, that are perfectly regular. It is a remarkable fact—which speaks in favour of the analogy which I have sought to establish between an hysterical cough and choreic convulsions—that, however continuous it may have been, it ceases entirely during sleep, and, as Dr. Lasègue justly remarks, this circumstance occurs frequently enough to acquire great diagnostic value.

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[1] "Archives générales de Médecine," 1854.

These attacks may recur somewhat periodically, and they may be excited, as well as suspended, by various circumstances which have no influence at all on a cough due to thoracic disease.

In some cases, which are, it is true, very exceptional, an hysterical cough has a peculiar *timbre*; it is hoarse, stridulous, and resembles a bird's cry; but one should be careful not to confound this cough, which even then retains some of its special characters, with the barking, the mewing, and the strange cries which are heard in hysterical cases, and which are related to the kind of tic of which I have already spoken. An hysterical cough is sometimes complicated with hoarseness and even with aphonia, sometimes also with obstinate vomiting, as in the case of a young person who came under my notice, and whose history I shall presently relate to you in a few words.

Dr. Lasègue makes the observation (in the excellent memoir from which I borrow a good deal of what I am now telling you), that "an hysterical cough not only remains identically the same throughout its course, but has no tendency also to assume other forms of hysteria; so that there are few instances of such a metamorphosis occurring." He cites, however, two cases which are exceptions to the rule, one of which occurred in Prof. Chomel's practice, and the other was observed in my wards by Dr. Lasègue himself, when he was my clinical assistant. The subject of the latter was a woman who, for the last three years, had been troubled with a cough which lasted almost continuously during several months of the year, recurring with less frequency in the intervals, and having all the characters which I have pointed out to you. She got rid of it after some deep emotion, followed by temporary loss of speech, and two days later by left hemiplegia, evidently of an hysterical nature, which got rapidly well without any treatment.

Such cases are not so rare as my learned friend thinks, for it would not be difficult to collect a pretty large number of instances, analogous to the one which Chomel published in the "Nouveau Journal de Médecine," for 1820, of paroxysms of an hysterical cough, alternating with convulsive seizures. I could myself cite several such; and many among you will surely remember having seen some of them; and only lately you could see a case of this kind in the wards under the care of Dr. Barth, my colleague in this hospital.

Lastly, you will find in one of the late numbers of the "Union Médicale," the case of a patient, under Dr. Hérard's care, whose hysterical cough was replaced, among other phenomena, by curious *sneezing*.

An hysterical cough may therefore alternate not only with the most common well-developed manifestations of the disease

on which it is itself dependent, such as convulsive seizures and attacks of hysterical paralysis, but it may also be replaced by local manifestations, such as vomiting and sneezing. What usually happens, however, is this, that the patient has previously exhibited, if not the marked symptoms of hysteria, at least, that group of special physical or mental dispositions which have been termed *hystericism* by some authors, and which consist in a *nervous changeability* carried to the highest point.

You know, gentlemen, what is meant by nervous changeability, namely, a condition intermediate between spasm and normal visceral innervation. It borders on the state of *vapours*, immediately precedes and is a necessary condition of that state, and only requires increased intensity of its phenomena, or the excitation of the slightest cause, in order to merge into it. Now this condition, which in most cases is only the highest degree of a predisposition to spasms, and enters into the constitution of many women, is most marked in those that are hysterical.

An hysterical cough generally sets in more or less suddenly, and, like all phenomena of a similar nature, without any appreciable cause. In the case of a young woman, who came under Dr. Lasègue's observation (the first of the examples which he has collected in his memoir), the hysterical cough came on after a simple cold which had lasted several days. The cold was perfectly well, and the catarrhal cough had ceased completely for the last eight days, when the hysterical cough commenced. You will certainly have an opportunity of seeing such cases. But although bronchitis may prove the exciting cause of an hysterical cough, the latter is by no means dependent on a peculiar predisposition to bronchial catarrhs ; and although, from its persistence and obstinacy, it often alarms the patient's friends and even her medical attendant, exciting in them fears that pulmonary phthisis is actually present or imminent at the very least, I never have seen this complaint begin with such symptoms.

In some cases, and always in profoundly hysterical women, a nervous cough sets in, in consequence of the presence of worms. I have already quoted the following instance, which Graves relates in his "Clinical Lectures." This illustrious physician was attending at Dublin, together with Sir Philip Crampton, a young lady who had lost all her strength from a spasmodic cough, which had lasted several months. Although no serious lesion could be discovered by auscultation, both these gentlemen could not help, however, believing in the existence of tubercles in the lungs, for there were fever and considerable emaciation. On the patient taking some turpentine, which an empirical old woman recommended, she passed a tapeworm, and the cough disappeared immediately, and her health was quickly re-established.

An hysterical cough is an essentially chronic complaint, lasting for months, and even years, uninfluenced by physiological phenomena, such as menstruation, which may occur while it lasts. Intercurrent febrile diseases suspend it, however, as they do hooping-cough. When it has persisted for a long time, it influences at last the patient's general health. The appetite diminishes or is lost, and digestion is impaired, especially if the cough be complicated with obstinate vomiting. The patient becomes pale and thin, complains of pain in the chest, and is unable to bear fatigue; fever is often lighted up; and you can understand how careful one must then be in order to recognise the nature of the case, and how he must have recourse to auscultation and percussion, in order to determine the absence of tubercles in the lungs, which suggest themselves to the mind from the first as the cause of the evil.

In spite of its persistence and obstinacy, and of the disturbances which it produces in the system, this singular neurosis almost never terminates fatally. After lasting more or less, it diminishes insensibly, and then disappears completely; in other cases it ceases suddenly, without any reason to account for this happy and abrupt termination. But whether it ceased by slow degrees or suddenly, the cure may be merely temporary. Like all hysterical manifestations, the cough may return, at the very moment when the patient thinks that she has got rid of it for ever; and, as on its first appearance, it comes on without any appreciable determining cause.

Of all the methods of *treatment* which have been tried against an hysterical cough, one alone has seemed efficacious to me, and I have rarely seen it fail, namely change of place; and the following case, to which I have already alluded, proves this most conclusively:—

A young lady, 17 years of age, whose health was habitually good, although she looked delicate, and who menstruated regularly, began to cough in May, 1852. Her mother was subject to spasmodic tic of the face, but she had never had any nervous attacks herself, although she had all the characters of an hysterical temperament. This cough attracted little notice for the first few days, but became so frequent that it alarmed her friends. It kept on all day, nearly without intermission, but ceased entirely when the patient slept in the daytime or at night. It was dry, sharp, stridulous, acute; audible from a pretty good distance, and recurring with a nearly unchanged rhythm. The most varied remedies—baths, cold affusions, antispasmodics, &c.—were tried, but without modifying its frequency or its characters. The breathing was such as to leave no doubt as to the regularity of the pulmonary functions; the fauces were neither red nor painful, and there was no alteration of the voice. This con-

dition lasted throughout the months of May and June; in the beginning of July some fever set in; digestion had already become laborious, and the appetite was nearly lost; vomiting came on, and the patient brought up her dinner half-an-hour after taking it, but not her morning meal. As her general health seemed to me to be rather seriously impaired, I recommended that she should be immediately sent to the South. My advice was acted upon; and on arriving at Orleans, after a three hours' journey, the patient, who felt fatigued, spent the night in an hotel. The vomiting ceased on that very day; the patient spent a good night, and had no fever; on the following day the cough ceased, and a complete cure ensued, which has lasted ever since. She remained away besides for several weeks.

A few years ago I saw, in consultation with my colleague, M. Guibout, a lady, 27 years old, who, for the last six months, has been suffering from a cough, having the peculiar rhythm which I have described to you. She had lost her appetite, had become anæmic and thin, and this alarmed her friends considerably: yet nothing abnormal could be detected on ausculting her chest with the greatest care. We prescribed a travelling-tour, and she got well immediately.

LECTURE XV.

SENILE TREMBLING AND PARALYSIS AGITANS.

Gentlemen,—I told you in our conferences on St. Vitus's dance that it could affect individuals of advanced age, although it most frequently attacked young adults, and I quoted in illustration a long and interesting case published by Dr. Henry Roger, the subject of which was a woman, 83 years old. This kind of chorea should not be confounded with another, namely, *chorea senilis* or *senile trembling*, as it is more appropriately termed, from which it differs totally, not only as to its nature, and the conditions which favour its development, but also as to the form which its symptoms assume, so that the two diseases may be easily distinguished from one another at first sight.

Senile trembling consists in a convulsive agitation of the muscles, produced by a series of involuntary but uniform contractions, taking place over a limited area, and following one another with excessive rapidity. At first generally confined to the extremities or to the muscles of the neck, it may spread to the whole of the body. It is most marked when the individual tries to execute voluntary movements, or when his mind is unusually stretched, or when he is under the influence of emotion. Rest and peace of mind diminish its violence or make it disappear entirely, while it ceases completely during sleep.

The causes of this complaint are unknown. It is usually said that this kind of trembling is a consequence of the weakness which old age brings on, but if this be true in some cases, it is not so generally speaking. For on the one hand, it is not invariably seen in very old people, and on the other hand, it pretty frequently affects individuals of middle age, and even young adults. You have yourselves known instances of this; and on this account, therefore, the term senile, when applied to this kind of trembling, is as inappropriate as when it is applied to gangrene due to the obliteration of an artery, and which may be seen at all ages, even in childhood.

However this may be, this kind of chorea is little known to pathologists, although it is pretty common. One point, however, is well known about it, namely, that it is incurable.

Senile trembling should not also be confounded with *paralysis agitans*, of which the woman now at No. 2, in St. Bernard Ward, presents us with an instance. She is a charwoman, aged 60; her complaint dates two years back, since which time, but especially for the last six months, she has complained of rapid loss of strength. Since then also she has been subject to trembling which, from being slight at first, became so violent that for the last four months she has been obliged to give up her usual occupation, from her inability to use her hands. The trembling has since become more general and involved the face, so that now her lower jaw shakes convulsively; and as she cannot shut her mouth, she dribbles constantly. She has retained all her faculties, and although she complains of the annoyance caused by this perpetual shaking which she cannot restrain, she does not speak of pain but only of a sense of extreme fatigue after the paroxysms of trembling. This is more marked on the right than on the left side, and when the strength of her right arm is tested with the dynamometer it is found to be equivalent to a power capable of raising a weight of 14 or 16 lbs., while on the left side, the instrument gives only 4 to 6 lbs. Cutaneous sensibility is unimpaired. In spite of this marked diminution of her muscular power, there is no paralysis properly so called, for when I try to flex or to extend her legs or her arms against her will, she resists me with an energy which I only overcome with some difficulty. I called attention, as you may remember, to the shape of her hands, for her four fingers deviate from their normal direction, and inclining towards the ulnar side of the limb, form with the forearm an angle of about 25°, so that the metacarpo-phalangeal articulation must, therefore, be partially dislocated.

Paralysis agitans, like senile trembling, is principally met with in persons of declining years, although it may affect adults, and I have seen a young man, 27 years of age, who was suffering from it.

In some cases it assumes another form, which it is important for you to know.

On October 16, 1863, I was consulted by an advocate, aged 58, of uncommon intelligence, and who for the last four years, after deep emotions, had been affected with the singular neurosis which I am going to describe to you, and which, in my opinion, was only a form of *paralysis agitans*. As he came up from the waiting into my consulting-room, he inclined his body forwards, hurrying his step, with his right arm, in a semiflexed position, resting against his body, and shaking very slightly. He sat down with some difficulty, and as if his trunk and legs were stiff. He then told me his story; how, in 1858, he had for more than a twelvemonth attended his wife assiduously, whom

he loved deeply and had lost. Grief and sleepless nights had exhausted him. He was then suffering from such nervous irritability that he could not bear to hear the ringing of bells; the least noise, the least annoyance, disturbed him beyond measure. He soon noticed that his arm seemed to shake slightly, and that the movements of the whole limb, but of the hand especially, became more and more difficult. In a short time, the leg on the same side became affected also, and his symptoms grew worse, without being in the least modified by any method of treatment. After a time, he had to give up writing, and when I saw him, he could sign his name with extreme slowness and difficulty only.

At first sight, he looks like a paralytic, but on examining him carefully, it is soon made out that there is only apparent paralysis, and that the case is a very curious one, which we cannot account for. For if I ask the patient to squeeze Burq's dynamometer, the instrument marks 100 lbs. much more than it does when I squeeze it myself. Squeezed by the patient's left or healthy hand, it marks 84 lbs. only, that is, 16 lbs. less than when the hand of the seemingly paralysed limb is used. If, when his arm is flexed, I try to extend it against his will, he resists me with extreme energy, and does the same when I attempt to flex, adduct, or abduct it against his will. There is no rigidity of the limb, and when the patient does not exert his will, his limb is perfectly supple and I can move it in every direction.

What takes place here then? The muscles have retained their strength, and yet their functions are nearly abolished. But let us try and analyze this curious phenomenon. When the will commands, the muscles obey instantly, and no appreciable interval intervenes between the act of willing and the muscular contraction. The movement may be repeated ten, fifteen, twenty, a hundred, or a thousand times in succession, as in the act of walking, for instance. If you suppose that in order to take two steps, the muscles have to expend an amount of strength equal to twenty pounds, if the same act be repeated a thousand times in an hour, a power of twenty thousand pounds shall be expended.

Now, let us see what occurs in the case of the patient whose history I have related to you. Let us suppose that he takes five hundred steps in an hour; each step shall have cost a 20-lb. power, and he will spend on the whole a force equal to ten thousand pounds, instead of twenty thousand, or, in other words, the motor power will be only one-half of the other. It was a very strange circumstance, that when I asked this patient to open and shut his hand as rapidly as he could, he moved at first quickly, then more slowly after scarcely a quarter of a

minute, and next he was unable to move at all. Just as a steam-engine, which is insufficiently heated, is unable to work continuously. But if the valves be closed for a moment, and the steam allowed to accumulate, the machine regains power for a time, but soon becomes powerless again after this artificial development of force. In the case of our patients, it would seem as if they could only spend a determinate quantity of nervous influence which is not reproduced in them with the same rapidity as in other men. They suffer then from a relative and momentary loss of power, but not from paralysis in the ordinary acceptation of the term.

The patient, whose history I have just related to you, was suffering from that form of the disease in which there is but slight shaking. The other woman, on the contrary, who was in bed No. 2, in St. Bernard Ward, presented considerable muscular agitation. In the man's case, the muscles were in a state of permanent contraction, and the sensation complained of was that of a continuous effort. In the woman's, on the contrary, although that sensation was complained of from time to time, there was more frequently muscular agitation. She stated that every paroxysm of shaking caused her as much fatigue as very violent exercise used formerly to do. By endeavouring to analyze these two muscular conditions, we shall understand better what occurs in what has been so inappropriately termed *paralysis agitans*.

All our muscles are in a state of relaxation during the period of rest. Their function ceases temporarily, and during that rest the aptitude which was lost or diminished from excessive action, is entirely regained. Suppose now, that in consequence of a modification of the nervous centres, the muscles should always be in a condition analogous to that of continuous effort, their excitability will be exhausted during their immobility, from the extensor and flexor muscles acting constantly and simultaneously. In the other form, the alternate rapid and involuntary movement of extension and flexion which constitutes trembling, expends the nerve force, as rigidity did in the former case, and power is wasted uselessly, at the expense of normal functions, so that when it becomes necessary to exhibit muscular power, the patient is incapable of doing it with the same continuity, or in the same degree, as before, and he will be in the same condition as an individual exhausted by extreme fatigue.

We meet with an analogous condition in those cases which I have termed *loss of muscular excitability*, a curious neurosis, of which I have seen very interesting instances.

A young lady, aged 18, and married for the last six months, came from Tours to Paris a few years ago, to be treated for

this strange neurosis. She was said to be paralysed. When I asked her to walk, she got up with determination, walked without staggering, and with perfect steadiness, ten, fifteen, twenty, twenty-five paces, then complained of feeling weak, and if no chair were near at hand, she was compelled to sit down on the floor. She lost all strength after this trifling exercise, and exhausted the amount of excitability possessed by her muscular nervous system. A few minutes' rest sufficed to give her back the aptitude which she had lost. In 1862 I saw another young lady in precisely the same condition. Mark that these two cases are only exaggerated instances of what we very frequently see. The power of restraining movements varies indefinitely, and we have no right to look upon these two cases as instances of paralysis, any more than we can pronounce those whose strength is exhausted after a moderate exercise lasting from ten to twenty-five minutes, to be suffering from paralysis.

It must be a well understood point then, gentlemen, that there is no *paralysis* at the commencement of this strange form of chorea, which is so inappropriately termed *paralysis agitans*, since there are cases (an instance of which came very recently under my observation) in which the muscular power, tested by the dynamometer, is, temporarily at least, greater on the shaking than on the opposite side. In the long run, however, real weakness supervenes, and towards the close of the disease the loss of muscular power is such that the existence of paralysis cannot be denied. Yet, it should be observed, that sensation is unimpaired.

The weakness of the genito-urinary organs is still more marked than that of the muscles. In males impotence sets in rapidly, and towards the last the urine is retained with difficulty, and there is sometimes incontinence, which may, however, be due to continued tonic contraction of the fibres of the bladder.

There may also occur another phenomenon which makes *paralysis agitans* resemble very much paralysis, due to hæmorrhage into, or softening of, the brain, namely, rigidity.

I was consulted, in 1863, by a superior naval officer, who for the last two years of a difficult command had been affected with *paralysis agitans*. At the end of a year he lost the power of writing, and when I saw him for the first time, the two last fingers of his right hand were firmly flexed into the palm of the hand, and it was only with slowness and with extreme difficulty that he could extend his thumb and his index and middle fingers.

Paralysis agitans, which some authors of eminence have confounded, not without some reason, perhaps, with *chorea festinans*,

is partial in the beginning, and may affect one arm alone, for instance. The limb shakes continually, and the patient complains of its feeling weak; this weakness, which is very slight at first, makes rapid progress; the corresponding leg shortly becomes affected in the same way, and involuntary convulsive movements show themselves simultaneously with a sense of diminution of muscular strength. The patient only hops when he tries to walk. As the disease progresses, it becomes general; the limbs of the opposite side are involved, and the patient's gait then becomes so characteristic, that his complaint can no longer be mistaken, although it cannot be satisfactorily described. His body inclines forwards as he walks, and he keeps the arm on the affected side in a semiflexed attitude, and closely pressed against the trunk. As his centre of gravity is thus displaced, he is obliged to run after himself, as it were, so that he keeps trotting and hopping on. He is unable to move without help, and in some cases, as he requires more assistance than is afforded by leaning on a stick, he can only walk by resting both his hands on the shoulders of an attendant, or supported from behind, otherwise he is sure to fall down.

I must add, however, that the complaint always occurs in paroxysms, and that after a paroxysm which may last from ten to forty minutes, and even more, the patient complains, not of pain, but of a sense of muscular fatigue, as after violent exercise.

When the disease becomes still more general, the muscles of the neck are convulsed, and the head then shakes continually; the muscles of the face are not spared, and as you saw in the case of the woman in my wards, the lower jaw drops, the mouth is always open, and allows the saliva to dribble out, which wets and messes the patient's clothes. Speech is, of course, embarrassed and indistinct. On the other hand, as the bladder gets paralysed, there supervenes retention, and subsequently incontinence of urine. All sexual power is lost. The convulsive movements are so often repeated, although they are not violent, that deformities result from them. Thus, from the patients' pressing against their hands constantly, their fingers get dislocated on the metacarpal bones, and their dorsal surface makes an angle with the back of the hand.

The intellect is at first unaffected, but gets weakened at last; the patient loses his memory, and his friends soon notice that his mind is not so clear as it was: precocious caducity sets in.

Paralysis agitans is an inexorable complaint which always terminates fatally within a shorter or longer period, in spite of all treatment. In three cases, however, which were under my observation until the end, I made the curious remark that death was caused by pneumonia. There is little probability

that other practitioners shall have an opportunity of noticing a similar coincidence between a neurosis and pneumonia.

I am not aware that the anatomical lesions special to *paralysis agitans* have been studied in France, and it seems that those who looked out for them, did not find any. We must pay great attention, however, to the alterations which Parkinson, Oppolzer, and Lebert have described; and allow me to quote a case, most carefully observed by Professor Oppolzer.

A man, aged 72, very thin and of very diminutive stature, was admitted into the "Clinique" on June 20, on account of a violent trembling which prevented him from using his hands. He gave the following account of the origin of his complaint: he had never had a serious illness until the age of 60, when during the bombardment of Vienna, in 1848, he happened by chance to get in the midst of the fight. He was struck with such terror, that he could not return home by himself, and had to be taken there. He had scarcely got over his fright, when a bomb burst near his house and alarmed him again. A few hours afterwards, on trying to take some food, he found himself perfectly unable to use his hands, because as soon as he tried to move them, they began immediately to tremble violently. He noticed also after a short time that his lower limbs trembled in the same manner, but less violently, so that he could still walk. The disease not only resisted all the measures employed against it, but also grew gradually worse. The trembling persisted even when he was at rest, and involved other muscles; lastly, paralysis was superadded to it. After a few years, he became incapable of standing erect, and as soon as he made the attempt, he had an irresistible tendency to fall forwards, so that in order to avoid falling down, he was obliged to lay hold of neighbouring objects, or to walk hurriedly. The keenness of his senses and of his intellectual faculties had diminished slowly but progressively.

The use of tea, of coffee, or of spirituous liquors always increased the trembling; and the agitation of the lower limbs was especially marked in the evening, when the patient had walked during the day. About six months ago, the sphincters, that of the bladder, in particular, became paralysed; the patient was then admitted into the general hospital on account of these complications, which seemed to improve at the end of a month. Five weeks ago, after a severe attack of vertigo, the patient dropped down suddenly, and was unable to rise, but never lost consciousness throughout. Since that time, the emaciation has increased very rapidly; the patient can stand and walk for a very short time only, and with very great efforts; and in addition, his articulation is embarrassed. He was in the following state when admitted into the Clinical Hospital: ema-

ciation very marked; earthy tint of the integuments, the surface of which is covered with numerous epithelial scales; the secretion of perspiration, which is increased on the face, seems on the contrary to be diminished in other regions of the body; the temperature of the skin seems to be lower than it normally is.

The muscles of the face, tongue, neck, and upper limbs are affected with violent trembling, which never ceases during the waking state, and is completely suspended only during profound sleep. The lower limbs shake periodically only, and when there is general exacerbation of all the symptoms. The muscles which are the seat of the trembling are *rigid at the same time*, especially the muscles of the neck and shoulders.

The pupils are equally dilated, and contract equally well under the influence of light. The mouth is only incompletely closed, and the saliva dribbles out of both corners over the chin. There seems to be no visceral lesion; there is merely slight dulness in front and at the back over the apex of the right lung. Auscultation detects besides, at those spots, a diminution of the respiratory murmur. The temporal arteries and the arteries of the limbs, especially the right brachial, are tortuous and rigid. Sensibility is normal everywhere; and the muscles contract, although somewhat feebly, under the influence of galvanic excitation.

The patient frequently complains of vertigo, and more rarely of cephalalgia. The stools are passed normally; the urine is alkaline and contains some pus. The patient answers very slowly but pretty clearly the questions which are put to him. His physiognomy is expressive of indifference and apathy. Treatment: carbonate of iron (a drachm for six doses to be taken in three days). The following is a summary of the further progress of the case:—From the 22nd to the 24th of June, a pretty severe diarrhœa set in, with involuntary stools, which yielded to the use of opiate injections. On the 24th, the carbonate of iron, which had been suspended during the presence of diarrhœa, is resumed.

June 25.—The patient slept only a little last night and was delirious: about ten in the morning, he had an epileptiform seizure, during which his head was pulled convulsively to the right, while his right eye turned outwards and upwards, and his left eye downwards and inwards. The eyelids and the tongue kept at the same time oscillating continually, while the muscles of the face were rigid and hard. The upper and lower limbs, on the contrary, remained flaccid, offering little resistance when moved about. The fit lasted about eight minutes, and during that time, the respiration and the pulse were weak and irregular, and there was complete loss of consciousness.

On the 1st and the 7th of July, fresh eclamptic seizures came on, after which, on each occasion, the trembling ceased for about half an hour, recurring after this with its former severity. General sensibility seemed to diminish from day to day, and the face had a stupid expression, reminding one of the physiognomy of individuals labouring under typhoid fever, in the second stage. The abdomen was swollen; there were involuntary stools, the urine contained some carbonate of ammonia and a few pus-cells as before; the patient lay in a sort of imperfect sleep, and it was almost impossible to fix his attention. He answered in monosyllables the questions that were put to him; his strength diminished rapidly, and pneumonia came on towards the close of his life. Death took place on July 11.

On making a post-mortem examination, several tubercular cavities were found at the apex of the right lung, and there was granular hepatization of the lower lobe of the same lung. Both ventricles of the heart were dilated and full of coagulated blood; their walls were discoloured and friable; the aortic valves were indurated at the base, the arch of the aorta dilated and ossified, the spleen of voluminous size, the mucous membrane of the bladder red, injected, and the muscular wall of the organ likewise injected. The other abdominal organs presented besides no other notable alteration.

The cranial bones were very thin, and their inner surface was rough. The dura-mater was thickened and adherent, here and there, to the inner table of the cranial vault; the pia-mater opaque and infiltrated with serosity: there was also a pretty large quantity of serosity in the sub-arachnoid cellular tissue. The cerebral convolutions were thinner, the sulci between them seemed deeper than usual, the cortical substance was of a pale brown colour, while the medullary was perfectly white, and traversed by dilated vessels; the cerebral substance was moist and of good consistency. The ventricles contained several drachms of transparent serosity, and the ependyma, principally on a level with the posterior cornu, was granular. In the substance of the right optic thalamus there was an apoplectic cyst of the size of a small bean, the walls of which contained pigment. The *pons varolii and the medulla oblongata were very manifestly indurated.* The spinal cord was firm, and the medullary substance of the lateral columns, principally in the lumbar region, presented opaque grey striæ. On making a microscopical examination, there was found *in the substance of the pons varolii and of the medulla oblongata an abnormal production of connective tissue*, accounting for the induration of those parts. *The opaque striæ in the lateral columns of the cord were due to the presence of connective tissue in process of development.*

In this case of Professor Oppolzer, then, gentlemen, the medulla

oblongata and the pons varolii were found indurated, while in the lateral columns of the cord, especially in the lumbar region, the medullary substance exhibited grey opaque striæ. All these changes, as well as the analogous ones noted by Parkinson and by Lebert, were the result of an hypertrophy of the connective tissue which enters into the composition of the nervous tissue. This hyper-formation had produced compression of the nervous elements, whence their atrophy and fatty degeneration. Such alterations, attended with induration of the affected parts, are termed *sclerosis*. In the cases in which dissection has shown incipient softening of the columns of the cord, in the same regions, this may perhaps have been a consequence merely of hyperæmia and vascular dilatation, which cause great modifications in the nutrition of nervous elements.[1]

Such alterations account for the powerlessness of treatment, for none as yet seems to have been attended with certain and continued success. I must mention, however, that Elliotson has ascribed the cure of a case of *paralysis agitans*, in a man 35 years of age, to the administration of carbonate of iron. But he admits that he was completely successful in one case only, and that no appreciable improvement was obtained in others.

Romberg tried the same treatment, and states that it failed; so that, although we may ascribe some part of the cure to the carbonate of iron in Dr. Elliotson's case, we may ascribe as great a share to the patient's age as to the medicine itself. Sulphur baths, iodide of potassium, and all powerful alterative remedies, should be tried, especially with the view of placing the patient in the most favourable condition for resisting the progress of the disease. Perhaps also, as in a case published by Dr. Axenfeld, the hyperæmic process which goes on might be arrested by revulsives applied to the upper portion of the vertebral column.

I have myself, in some cases, obtained good results from the use of large doses of spirits of turpentine, and from hydropathy; but I have not cured a single patient: and this sad complaint is, in my opinion, as intractable as progressive locomotor ataxy.[2]

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[1] [Petræus (quoted by Dr. Handfield Jones, in "Funct. Nerv. Dis.," p. 266) has recorded a severe case of paralysis agitans, observed in the Copenhagen hospital, which proved fatal. Nothing was found at the autopsy but fatty degeneration of the heart, and pneumonic consolidation of the right lung.—Ed.]

[2] [There are a few instances on record in which paralysis agitans has been cured; and, although there is no doubt that many cases of chorea, or of mere tremor, have been mistaken for this affection, yet some of these examples of cure have been published by such competent observers that no doubt can be entertained as to their authenticity, and as to the real nature of the case. The following, which was kindly communicated to me by my friend and colleague, Dr. Ramskill, seems to have been one of those rare triumphs of medical art over this generally intractable disease:—

A carpenter, aged 52, who had always enjoyed good health, and had never

had gout, syphilis, or rheumatism, and had always been free from extraordinary anxieties, came under the care of Dr. Ramskill, suffering from paralysis agitans. He acknowledged having been rather intemperate. Both his parents died of old age. The complaint began two-and-a-half years previously, by trembling in the right hand, which soon extended to the whole arm, and then to the leg of the same side. Two years afterwards, the opposite side suffered in a like manner, and when he presented himself to Dr. Ramskill, he was incapable of dressing himself, and was obliged to run when he attempted to walk. He had some difficulty of articulation; but otherwise, his general health was very good. Various remedies were tried without any good result. On October 8, 1864, he was ordered cod-liver oil (ʒj), with phosphorized oil of the Prussian pharmacopœia (♏ iv.) ter die. He shortly began to show signs of amendment, and by July 3, 1865, he had grown stout, the trembling had entirely ceased, his gait was natural, although brisk movement occasioned an effort.

Dr. Russell Reynolds has published the case of a man, aged 57, who applied to him on account of vertigo and general disturbance with paralytic tremor of the right upper limb, which in the situation of the biceps was 4° F. hotter than the other. The sensibility was unaffected. After five applications of an 120-link Pulvermacher's chain, each lasting an hour, the spontaneous jactitation completely ceased. By continuing the same treatment every other day, the power of the arm was almost quite restored in a month. The disease was recent, having commenced only fifteen days before he was submitted to treatment.

Dr. Handfield Jones (in "Funct. Nerv. Dis.," p. 267), expresses the belief that there exists a functional nervous affection which at least bears a very close resemblance to paralysis agitans, and between which and chorea there is no very material difference. It seems probable, in his opinion, that the disorder depends, in some instances, at least, on increased excitability of the nervous centres, of such a quality that it will not tolerate tonics, and requires rather calmants. In support of this view, he relates the case of a labourer, æt. 47, a large man, who complained of tremor of the right upper limb, the muscles of which had full power, but quivered and twitched constantly, except when he was quiet in bed: the pectoral muscles of that side were involved. There was some tremor of the legs, but nothing like that of the arms; both, however, were decidedly weak, and the knees were sometimes very stiff. Strychnia, iron and ether, and faradization failed; nay more, this treatment, and especially electricity, was rather injurious than otherwise. Under the influence of tinct. hyoscyami, ʒss, ter die, he rapidly improved, and ceased attendance in a fortnight. Two years previously, this man had been under Dr. Jones's care for the same affection, had soon got well, and remained quite well for one year, after which time the disorder came on again at intervals, on some days and not on others; so that it may be questioned whether the rapid improvement, on the second occasion, may be termed a cure?—Ed.]

LECTURE XVI.

CEREBRAL FEVER.

Instances of different forms of Cerebral Fever.—Description of the disease: three stages which are generally pretty distinct.—*Premonitory stage,* characterised by a group of general phenomena, which may be seen in other diseases, but which are never so marked and never so prolonged as in this complaint.—*Second stage:* absence of fever; the pulse becomes remarkably slow, and the breathing peculiarly irregular.—This irregularity of the respiratory movements is a sign of great value.—Differential diagnosis between cerebral fever and typhoid fever.—*Third period:* the pulse quickens again, and often to an extraordinary degree.—Prostration, delirium; convulsions, at first partial, then general; paralysis.—Cerebral fever is nearly always, not to say always, fatal, whatever be the *treatment* adopted.—The post-mortem appearances are more indicative of cerebro-meningitis than of meningitis.—Whether tubercular or not, the complaint runs the same course.—Chronic hydrocephalus.—It is not a consequence of cerebral fever.

Gentlemen,—At No. 33, in St. Bernard Ward, there died a young woman, 23 years old, who had been admitted on March 13, 1866, on account of paralysis of the right limbs, without implication of the face, and due to *cervical arthritis,* marked externally by great swelling of the first vertebræ, and by pain exaggerated by the least movement of the head, which, on that account, the patient kept perfectly motionless.

The paralysis had supervened under the following circumstances. The patient stated that she had in general enjoyed good health, although she was of a delicate constitution. Eighteen months before she was admitted into the Hôtel Dieu she was seized with pain in the neck, acute enough to prevent her from turning her head, especially to the right. She had at the same time a sensation of constriction and stiffness in that region, which was markedly swollen. Ointments (the composition of which she could not tell us), poultices, and subsequently the application of leeches, did not arrest the progress of the disease. Within ten months the complaint had made such advances that the poor patient could no longer lie with her head on the pillow, as the pressure exaggerated her pains, which were much worse on the right side of the neck. She complained at the same time of a sense of constant numbness in that part. The phenomena of the disease soon assumed a more complicated form, and fifteen months after their invasion she complained of a diminution

of strength in the right arm and leg. This weakness went on increasing, and in a month's time passed into paralysis, which never was complete, however. The patient could still walk, although she could only raise her leg with difficulty, and dragged it; she had not lost entirely the power of moving her arm, although she could not use her hand to do her customary work, not even to carry her food to her mouth. Formication, followed by numbness, preceded and accompanied the paralysis of motion, and was the only disorder of sensibility, which was, in other respects, perfectly normal everywhere. There was no impairment of the intellect; the special senses were normal, and there had been at no time the least febrile reaction. For the last two months or so the appetite alone had failed, and the patient ascribed it to her being unable to take as much exercise as before. Her digestion was perfectly regular, notwithstanding.

From the first day that I saw her, I easily made out that the hemiplegia was due to disease of the vertebræ. My attention was attracted by the swelling of the neck, which was much larger superiorly on the right, especially on a level with the two first cervical vertebræ. The swollen part was painful, and the least movement of the head, either when the patient herself attempted to raise or turn it, or when I tried to move it with great caution and slowness, was attended with acute suffering.

The case was evidently one of white swelling of the atloido-axoidean articulation; and although auscultation of the chest revealed no signs of pulmonary tuberculisation, and the patient declared that she was not liable to colds, and that there was no tendency to phthisis in her family, I could not but diagnose scrofulous disease of the vertebral column. Although I could not discover any trace of syphilitic diathesis, still suspecting that the disease might arise from constitutional syphilis, I prescribed mercury (corrosive sublimate baths and calomel in divided doses); but as salivation was soon induced, I suspended the calomel.

The disease continued to make progress. In order to calm the pain, which had become more intense, I ordered poultices, made with powdered conium leaves, and kept on the neck day and night. The pain still went on increasing, and it was not only felt in the head by July 17, but in the legs also, the hypogastrium and the groins. As menstruation (which had been regular until eight months ago) had been suppressed at that time, I thought that the pain might be due to a tendency to a re-establishment of that function, but vomiting having set in, I began to fear that it might announce the invasion of a cerebral affection. Indeed, in the course of the day the patient, whose mind was perfectly clear, began to exhibit some embarrassment of speech. The pain in the neck grew much worse, the paralysis

of the limbs became more marked, and by the next day the expression of the face had altered appreciably.

The disease remained stationary until the 23rd: calomel, which had been resumed on the 18th, was continued in the same divided doses. The drug had no apparent effect on the digestive tube, and the stools were regular as usual. On July 23, we found strabismus, and for several days previously the patient had complained of *seeing double*. On the 24th, deafness came on; the patient had an attack of syncope during the night, and on the next day I found her in a feverish state, with a hot skin, and the pulse beating at the rate of 120 in the minute. The abdominal walls were retracted and boat-shaped; the *cerebral macula* was produced with the greatest facility, and persisted for a long time. The patient had fits of *absence* during the day, and did not know the persons about her; delirium set in during the night, but disappeared in the morning. The strabismus, and the changes in the expression and the colour of the face, which was alternately very red and of a deadly pallor, became more and more characteristic, and in the evening, the stools were passed involuntarily.

The symptoms grew worse and worse. The respiration became very irregular, from four to five or eight inspiratory acts following one another with extreme rapidity, and being then followed by a considerable pause. There was extreme vascularity of the skin, and the cerebral macula was brought out by the least friction; the strabismus was pushed to its extremest limits, and the pupils were dilated. The intellect was still pretty clear, and the patient answered questions, but without separating her teeth, her jaws being firmly closed. Death took place on July 28, at 4 P.M.

You remember, gentlemen, what we found on examining the body. I had, during life, diagnosed white swelling of a vertebral joint as the starting point of a cerebro-meningitis of the base, and, indeed, we found traces of a violent inflammation of the pia-mater, which was infiltrated with pus, and covered as with a greenish transparent veil the annular protuberance and the space between it and the optic commissure. The fissure of Sylvius was filled with a sero-fibrinous material. On making sections of the brain, the fornix and the septum lucidum were found in a pulpy condition; the lateral ventricles contained a notable quantity of serosity, and their posterior part was softened as well as the corpus callosum. There were no tubercles, and no granulations anywhere. The spinal meninges were injected, and the cord itself was softened on a level with the articulation of the atlas with the axis, while those vertebræ, which were markedly larger on the right than on the left side, exhibited all the characters of osteitis. Their articular surfaces and that of

the odontoid process were deprived of cartilage, roughened, and pierced with numerous foramina, but they contained no tubercular matter, either collected in masses, or in a state of infiltration. The cellular tissue in the neighbourhood was infiltrated with plastic lymph and with pus.

The lungs looked healthy, and showed no traces of tubercular deposit.

About the same time as this patient was admitted into St. Bernard ward, another young woman died there also, but much more rapidly, of cerebral fever, which had supervened under different circumstances. She came to the hospital during the day, stating that she had been unwell for the last nine or ten days. She gave a pretty good account of her sensations, but seemed by no means uneasy about them; she laughed at and joked about her own condition (note this well, gentlemen). Yet, I was far from being satisfied with her state. I noticed that her face was flushed, her aspect dull, her pupils dilated, and that her left limbs were somewhat weaker than the right ones; the cerebral macula was produced with the greatest facility. When my clinical assistant saw her in the evening, he diagnosed encephalitis, and I made the same diagnosis the next morning. Three days afterwards the patient died. She had conversed with me very pertinently on that very same morning, and had even joked, but an hour after my visit, she fell into a profound stupor, and died suddenly.

Dissection disclosed on the surface of the brain, at its upper and under aspects, the presence of granulations in the meninges, and a small mass of tubercle at the base. The corpus callosum was completely softened, and reduced to a pulpy condition, as well as the posterior part of the walls of the lateral ventricles, the cavity of which contained some serosity. The septum lucidum and the fornix were also softened.

Some of you may also recollect the history of a third patient, a male, who died of cerebral fever about the same time as these two women. He occupied bed No. 19, in St. Agnes Ward. He was 21 years old, and had been seized, about eighteen months previously, with rheumatic pain in the left leg, which resisted all treatment. Two months before the complaint, of which he afterwards died, set in, he came to Paris and took a situation as shop messenger. He worked beyond his strength at that place, he says, and a fortnight before his admission into the hospital he complained of a violent pain in the head, which set in suddenly. He went on working as usual; but he felt so exhausted every evening that he could scarcely find strength to get home. Three or four days went by. His appetite had failed sensibly for about a month, and since he had come to Paris he had been subject to diarrhœa, passing two or three liquid stools in the

twenty-four hours. During the above three or four days he had lost his appetite completely, and he soon was compelled to give up work. His headache increased markedly in violence, especially across the forehead, at which part he complained of continued, unbearable throbbings, giving him the sensation as if his skull was going to burst. He had pain in the eyes also; and did not sleep at night. From the beginning, he had had during the day very copious vomiting, and could keep no liquid on his stomach. The matters which he vomited contained bile; and he complained of a bitter taste in his mouth. The tongue was coated with a thin, whitish fur; the skin was not abnormally hot, but the slowness of the pulse (which beat 25 times in the minute) coinciding with intense cephalalgia, sleeplessness, and dilatation of the pupils, made me anxious.

Constipation had replaced the diarrhœa, and in order to produce revulsion towards the lower part of the large intestine, I prescribed a purgative (calomel and jalap).

On the next day the pulse was slower, 46; the vomiting was less frequent, but the cephalalgia being still more violent, if possible, I tried to relieve it by the application on the forehead of compresses steeped in a solution of cyanide of potassium (20 grains to 3 ounces of distilled water). The pain began to diminish forty-eight hours afterwards; but the patient had complained for the last three days already of some disturbance of vision; his eyes looked like those of a drunken man; the pupils were not dilated to an extraordinary degree, but contracted badly under the influence of light. Lastly, the cerebral macula was easily produced. In the course of that evening (the fifth day after his admission) he was found in a very prostrate condition, with staring eyes, and a stupid look, apparently insensible to everything around him, and picking the bedclothes. His skin was hot, but his pulse was not more than 64. He had an attack of syncope some time afterwards, and during the night he uttered plaintive cries without coming out of his somnolent state. The sopor was more marked the next morning, and his eyes remained half-closed without the pupils being dilated. The breathing was uneven, and the patient uttered plaintive cries again, as during the night. Although apparently insensible to everything around him, he felt very well when he was pinched, and withdrew his arms. The carphology persisted, the fever was more intense than on the preceding day, and yet the pulse was not more than 84 or 88. There was again very obstinate constipation, so that I ordered an enema to be given (an ounce of decoction of senna leaves, and half-an-ounce of sulphate of soda). This produced very slight effects. On the 18th, in the morning, profound coma had succeeded to the somnolence; the pulse was small, and 140

in the minute; there was left hemiplegia. On the right side sensation was still retained, for when pinched the patient withdrew his arm and leg, while on the left, pinching was not felt. The bladder was distended.

Death took place at 4 A.M. We had already ascertained that two brothers of this patient had died at the same age and in the same manner.

The autopsy showed the presence of encephalitis. In the posterior part of the right optic thalamus there was found an indurated mass, of a yellow colour, and dotted with numerous red points (capillary hœmorrhage). In the centre of this mass were other small nuclei, not larger than millet-seeds and having all the characters of tubercular matter. The cerebral tissue was softened, but not diffluent around the whole mass. The lateral ventricles contained about a teaspoonful of reddish serosity, and small grey granulations were scattered over the meninges, which were very dry.

The two layers of the pleuræ adhered firmly to one another, and in the substance of the lungs, which were congested, a few small tubercles were scattered.

I wished to recall these cases to your memory, gentlemen, before speaking to you of cerebral fever *à propos* of two babies, one of whom died a few days ago, and the other only yesterday, so that I shall have an opportunity of showing you once more the characteristic lesions of this cruel and inexorable complaint.

The first of these children was a little boy ten months old. Nine weeks previously his mother had brought him to me for the first time, on account of an unhealthy looking ulcer which he had in the neck, and which was covered with pultaceous concretions. The perpendicular and indurated edges of the ulcer, its uneven and hard bottom, and its colour, had all the appearances of a scrofulous ulcer. I had it painted with tincture of iodine, and three weeks afterwards, the surface of the ulceration had been modified, a complete cure was brought about, and the baby was discharged from the Hôtel Dieu. I had, however, been struck with the patience with which the child bore the pain produced by the iodine paint, which is generally very acute when applied to a raw surface; but this baby evinced very little sensibility. I was surprised at this, and wondered whether something serious was not hidden under it. The cause soon became apparent, for my fears were realised in a short time. A fortnight after he had been discharged, the child was brought back, suffering from cerebral fever which was incubating during his first stay in the hospital. The development and evolution of this fever were so regular, so classical (if I may be allowed the expression), that there could be no doubt

as to the nature of the case, although, in too many instances, the deceptive course of the disease misleads men of the most consummate experience.

The child's mother gave us the following statement as to the manner of invasion of the complaint. She brought the child back on a Monday; eleven days previously she had, on her own authority, given him some ipecacuanha on account of a cold in the head. The ipecacuanha brought on vomiting, which had not ceased even when I saw the child; he was strangely agitated, had no sleep, but merely dozed, rousing himself at intervals, and uttering a loud cry.

These symptoms, namely, vomiting, insomnia, somnolence, with sudden awakenings and utterance of loud cries, too clearly indicated incipient brain-fever. The pulse gave no indication yet, but in another week, its inequality, and the diminution in the number of pulsations, became a new feature of the disease. Yet the child continued to take the breast. As the vomiting had ceased, one who was not forewarned might have thought that the child was better. But, independently of the signs which I have mentioned, and which left no doubt on my mind, I already noticed that the child was singularly agitated when I came near him, but soon became calm again, and fell into a doze. This was a symptom of considerable significance; and all the others showed themselves in succession, namely, cerebral macula, dilatation of the pupils, paralysis more marked on one side of the body than on the other; lastly, convulsions, and extraordinary frequency of the pulse, which from 68 rose to 80, 100, 140, 160, up to 208 on the day preceding death.

On making the autopsy, I found notable thickening of the meninges which, about the optic commissure and in the fissure of Sylvius, were infiltrated with fibroplastic elements and concrete albumen, while there were numerous granulations disseminated on the surface, especially over the left cerebral hemisphere. The septum lucidum was in a perfectly pulpy condition; the fornix was less softened, but tore on the least pulling, and the softening had also involved the posterior wall of the lateral ventricles.

There were granulations in the lungs also, while the bronchial glands were converted into tubercular masses, and similar ones were found in the spleen.

The other child, who died yesterday, and whose body I am going to examine in your presence, was a little girl eighteen months old, nursed by her own mother. Although of an apparently sound constitution, she had been seized about six weeks ago, when she was noticed to have an unusual, sad look. This could not be ascribed to the process of teething, because she had cut her first group of teeth for the last four months,

and there was no indication that the evolution of the upper incisors, which were to form the second group, had commenced. Sadness setting in unaccountably, is a premonitory sign of great value in a child; it points to a condition of *malaise*, surprises the child's friends, makes them uneasy, and is often mentioned by them, as it was in this case by the mother of the little girl. She added, besides, that the child's sleep was not continuous, and was, as it were, disturbed; yet a symptom which is very common at the onset of cerebral fever was absent in this case—the child did not start out of her sleep and did not utter the peculiar cries noted in the case of the little boy which I related to you just now, and which constitute a sign of some value in the history of cerebro-meningitis. Vomiting set in a week ago: the child brought up everything that she took, panadas, her mother's milk, sugared drinks, so that her mother began to feel seriously uneasy. These fears increased three days later on account of another symptom which she described very well and which it is essential I should point out to you. The child cried whenever she was taken up, as if in great pain; and, indeed, there was *general hyperæsthesia*. Lastly, four days ago, convulsions came on, at first on the right, then on the left side; and it was then that the mother came to the hospital.

Let us now rapidly review the symptoms which this child presented, and compare them with one another, as well as with those which are common to brain-fever and other diseases.

When I first saw the child, I was struck with the motor disorders of her visual organs. There was very marked convergent *strabismus* of the right eye, the pupil of which was dilated, although less notably than that of the left eye; consequently, the muscle supplied by the sixth nerve must have been paralysed. Sight seemed to be abolished on the left side, because when I held my finger in front of that eye, there was no longer the involuntary and instinctive working which usually occurs for the protection of the threatened eyeball. There was probably blindness, or at least, a very marked diminution of sight. More or less complete amaurosis is a symptom which you have noted yourselves in all our cases of brain-fever, and which is complained of also by children old enough to talk and give an account of their sensations. In the little girl in question, the greater dilatation of the pupils, the absence of all movement of the eyelids, the strabismus of the right eye, very clearly indicated that sight was impaired.

The head was slightly pulled back also, the left arm was stiff, and was from time to time the seat of clonic movements of flexion and extension. The thumb of the left hand, forcibly adducted into the palm, was covered over by the fingers, which were like itself convulsively bent; when an attempt was made

to stretch them out, they yielded with some facility. On exposing the child's abdomen, it was seen to be excavated—hollowed out like a boat from the sinking in of its walls. This sign is of great value in the history of cerebral fever, because it is nearly constant. In a great many cases it may help to distinguish the brain-symptoms of cerebro-meningitis from those which appear secondarily in the course of other diseases, such as typhoid fever, for example. You must not think, however, that there is no chance of error when this symptom exists; its diagnostic significance, although of great value, is not always absolute, and not long ago I found among my papers notes of a case which shows how difficult it is in some cases for a medical man to decide.

The subject of that case was a little girl seven years and a half old, who was under my care at the Children's Hospital, towards the close of the year 1852. She was of a lymphatic constitution, and had had for months past a cough, and some diarrhœa. She had been worse for two or three days, and had been seized with vomiting. She was delirious the night after her admission, and on the next morning she was very prostrate, although conscious. Her pupils were dilated, more so on the right than on the left; her belly was retracted in the manner I have described above, and was tender on pressure. Her pulse was excessively slow, 56 in the minute. (I insist on this fact, which is almost constant in brain-fever.) In addition, the *meningeal* or *cerebral macula* (which I will presently describe more particularly), was easily produced, and became still more marked on the following days. She never uttered, it is true, the hydrocephalic cry, nor was her breathing unequal; but with these exceptions, all her symptoms seemed to point to cerebro-meningitis. Yet, the case was one of typhoid fever, and after death I found no changes in the brain and its meninges, while the swelling and ulceration of Peyer's patches, in the small intestines, were characteristic of typhoid fever.

Dilatation of the pupils, even when it is not equal on both sides, retraction of the abdominal walls, constipation (for the girl's bowels from being loose had become costive), and the cerebral macula itself, are not therefore absolute pathognomonic signs, although they are phenomena of very great value.

Now, what are the characters of that *cerebral* or *meningeal macula* which I have taken care to point out to you in the above cases, and which you always see me carefully look for in individuals whom I suspect of being the subjects of cerebro-meningitis? When, in order to ascertain how many teeth the little girl in St. Bernard ward had cut, I opened her mouth with my hands, you must have been struck with the bright red tint which her skin immediately assumed. Again, when I

very gently made on her abdomen with my nail cross markings, longitudinally and transversely, in less than half a minute the portion of skin which I had touched was suffused with a very bright red tint, which was diffused at first, but grew by degrees fainter, leaving along the track where the nail had passed, lines of a deeper red colour, which persisted for a pretty long time. This is what I mean by *cerebral macula*. I was the first to call attention to it more than twenty years ago, and I then called it *meningeal* macula. This singular phenomenon, which can only be explained by a deep modification in the vascularity of the skin, is a sign of sufficiently great importance to arrest our attention for a while, although I repeat, it is not of absolute value when the differential diagnosis of cerebral fever has to be made.

The regions where the macula appears most easily are at first, and above all, the anterior aspect of the thighs, the abdomen, and the face. I have just described its characters. If after exposing the patient, his skin be gently rubbed with a hard body, such as a pencil, or simply with the nail, the part touched rapidly becomes of a bright red colour, which persists for a more or less prolonged period, eight, ten, or fifteen minutes. Its existence has not been denied (for it is unquestionably brought out under those conditions), but the importance which I attach to it has been questioned, on the ground that it was met with in other diseases than cerebral fever. I admit that this may occur, and the case which I related to you just now proves it; but whereas it is an invariable, constant phenomenon in cerebral fever, observed throughout the whole course of the disease nearly, from the beginning to the end, it only appears exceptionally and accidentally in other affections. It has been said that this mottling was always observed, when looked for, in children suffering from simple febricula. But I protest against this assertion, gentlemen; and I have more than once shown you in our clinical wards young children labouring under intense fever, attending sometimes violent stomatitis, and at other times grave pulmonary catarrh, or grave pneumonia, and when I have in such cases tried to produce the mottling by rubbing the skin, and even so roughly as to scratch it, I indeed made the parts which I touched red, but the redness was never to be compared, as regards intensity and duration, with the redness produced in individuals suffering from brain-fever, even by the gentlest frictions. In the latter, it persisted for a good while; and it not only involved the parts which had been directly touched, but spread also for several centimètres beyond them, while in other complaints it is exclusively limited to the points where it had developed itself. I lay so much stress on this point, because it is, in my opinion, of great significance in

a good many cases, when the possible confusion between brain-fever and other diseases has to be avoided, such as typhoid fever, attended with cerebral phenomena or convulsions, either idiopathic, or occurring at the outset of exanthematous fevers, or grave pulmonary or other inflammations. The mottling is almost never produced in eclampsia; and when it occurs in typhoid fever, as in the instance I mentioned to you, it rarely has the same intensity and persistence, and rarely shows itself at all stages of the fever.

From what I have said, it follows, therefore, that there is, properly speaking, no one invariable pathognomonic sign of cerebral fever. But in this, as in all clinical questions besides, it is not isolated symptoms, but groups of symptoms, the manner in which they appear and are evolved, and their mutual relations, which characterise the disease. We must not look at a portion of the picture only, but at the whole at once; in order to know the drama well, the whole play must be seen, and not one scene alone. Yet, in order to write the history of the disease, we are obliged to analyse its symptoms, and to make divisions with the view of facilitating description. I will, therefore, speak of cerebral fever as having three stages, which, although they are far from being constantly present, and from being always perfectly distinct from one another, are yet sufficiently distinguishable by certain predominating symptoms. The first of these, the *premonitory stage*, is of great importance. Rilliet (of Geneva) the joint author with my esteemed colleague, Dr. Barthez, of a work on diseases of children, has laid most stress on this point, and he has recorded a pretty good number of cases which came under his own observation, and in which he was enabled to foretell the more or less immediate invasion of brain-fever by means of certain signs which I am going to enumerate to you.

A *change in the child's manner* in a great many cases, but not in all, is a sign that brain-fever is imminent. This change shows itself for a more or less prolonged period, for four or six weeks, or for two, three, and more months sometimes, before the complaint actually sets in. The child is unaccountably sad, and takes less pleasure than usual in his games; his temper becomes sour, and he shows himself more irritable towards his parents, his brothers, and his companions. There is at the same time (and this is a valuable sign) marked *emaciation*. Sometimes there is bilious *vomiting* which cannot be accounted for, and which recurs at more or less distant intervals. Sleep is not so profound as it used to be, and may even be replaced by complete watchfulness; in some cases this imperfect sleep is agitated, disturbed by painful dreams, by sudden starts, accompanied by those characteristic cries which become subsequently

more frequent, and of which I shall speak more particularly by and by. Rilliet ascribes this series of symptoms to already existing lesions, more especially to cerebral lesions, which, although latent and assuming a chronic or, at the most, a subacute course, still exercise from that very period an injurious influence on the organic functions—those of the brain chiefly. As in children who die of brain-fever, tubercles are almost invariably found—not in the viscera themselves, but in the bronchial or the mesenteric, or, more rarely, in the cervical glands, it is conceivable how a tubercular affection may give rise to the general disorders which I have mentioned, and how more or less marked emaciation may result from it. As to the brain symptoms, the change of temper, the watchfulness, or the disturbed, interrupted sleep, the cries uttered by the child, apparently indicating a sharp pain in the head, they are accounted for, according to Rilliet, by the brain-lesions which are nearly always met with when a *post-mortem* examination is made. These lesions consist in granulations scattered in the meninges over the surface of the brain, and in the Sylvian fissure, and which have been shown by the microscope to be of a tubercular nature. We may imagine, therefore, what an injurious influence the morbid process which precedes and accompanies the evolution of these morbid products, however slow it may be, exercises on the functions of the central apparatus of innervation.

I do not deny, gentlemen, that these premonitory symptoms occur more frequently at the outset of brain-fever than of any other complaint; but one would exaggerate their import, if he were to regard them, as Rilliet has done, as characteristic of cerebral fever exclusively. They, indeed, seem to me to depend much less on an actual lesion than on the general condition which in this case passes into cerebro-meningitis, but in other cases, into latent pleurisy, or into pulmonary tuberculisation, or at least into tuberculisation of the bronchial glands, and in other instances, again, into *tabes mesenterica*, namely, tuberculisation of the peritoneum and tubercular infiltration of the mesenteric glands.

The premonitory symptoms indicate, therefore, the imminence of some disease rather than an actual disease. We know how the temper of a child changes under the influence of the least *malaise*, and such changes are, besides, common enough in adults, and there are very few among us who have not experienced them even in slight indispositions. They strike all the more in children, and occur all the more easily that their temper is more mobile. There is no need, therefore, in order to explain the sadness and surliness of individuals threatened with brain-fever, or their unusual repugnance to join in games of

children of their age, to appeal to the presence of a brain lesion, when such morbid phenomena are accounted for by the *malaise* resulting from the deep perturbation of the functions of the whole system, caused by the slow and fatal manifestation of the tubercular diathesis.

Although these morbid phenomena may usher in other affections, it must be, nevertheless, admitted that they are never so marked as in the prodromic stage of cerebral fever; and there is one point concerning them to which I must particularly call your attention. You may have observed these premonitory symptoms in a child who is scrofulous, or who is the issue of phthisical parents, and have either imparted your fears to the friends, or kept them to yourself, when you see the child suddenly regain his former cheerfulness and be restored to health, with the exception of some loss of flesh; then, the symptoms recur and disappear again until the day when the disease breaks out. I perfectly remember the case of a little boy whom I saw in the Tours Hospital, when I was a medical student. He was from time to time seized with fearful pains in the head, with vomiting, somnolence, slowness of the pulse, &c. These symptoms lasted for three or four days, and on every occasion, Bretonneau diagnosed the approach of cerebral fever; but the storm blew over. At last, one day the symptoms did not intermit, and we had occasion to witness all the scenes of the sad drama of tubercular cerebro-meningitis. Dissection disclosed, in addition to the ordinary lesions of brain-fever, the presence of a large tubercular mass in the convolutions of the cerebellum, with softening of the surrounding tissue. It rarely happens, indeed, that in such cases the symptoms be not dependent on the presence of some organic brain lesion, and particularly on tubercular deposit. In such cases, independently of the symptoms which I have already mentioned, intermittent cephalalgia, convulsions, and partial paralysis may supervene at more or less distant intervals, until brain-fever sets in, which rapidly draws to a fatal termination.

Whenever, therefore, the above group of morbid phenomena are found to exist, the practitioner should be on his guard, especially if the family history of the patient points to a tubercular diathesis, because he may soon witness the characteristic phenomena of the *invasion* of brain-fever. In general, *vomiting*, of an obstinate character, opens the scene; very often, this does not excite much anxiety at first; it is ascribed to a trifling indisposition, and as a moment before it set in the child seemed to enjoy his usual state of health, and ate with some appetite, it is put down to indigestion. This opinion is retained for a day or two; but as the vomiting persists and recurs frequently, alarm is excited. This symptom, namely, persistent

vomiting, is of primary importance, and whenever it shows itself without attendant fever, in a child who has been vaccinated and who has already had exanthematous fevers, brain-fever should be suspected.

There is, in general, *constipation* also.

Persistent vomiting and constipation are already two symptoms of great value. The patient complains at the same time of intense *cephalalgia*, which is usually general, although more acute across the forehead and sometimes at the vertex. This symptom alarms the friends most, and is the one to which they call the practitioner's attention. This headache is not by itself, however, a sufficiently characteristic sign, for there are many other diseases which set in with a more or less violent cephalalgia, proportionate to the intensity of the febrile reaction of which it is an epi-phenomenon.

Its persistence, however, and that of the vomiting, are all the more peculiar in cerebral fever, that the *initial fever* of the disease does not run the same course as in other affections. Thus, it consists of several paroxysms, instead of a single one. The patient has two or three rigors in the course of the twenty-four hours, and after each rigor some heat of skin and perspiration; sometimes, this rigor recurs several days in succession, at the same hour; in other very rare instances, the fever is continuous but is moderate, with frequent remissions. Thus, febrile action running a peculiar course, violent cephalalgia, more or less limited to a portion of the head, constipation, obstinate vomiting, interrupted sleep, or complete wakefulness, alteration of temper; such are the symptoms of the first stage of cerebral fever, to which are pretty frequently superadded singular perversions of sight, amblyopia, hemiopia, and strabismus.

I have often related the following cases, which are so interesting that they are deeply impressed on my memory. About twenty years ago, I saw with my excellent friend, Dr. Pidoux, a girl 6 years old, affected with cerebral fever. She had usually a very strange temper, and although her mother was full of kindness and indulgence for her, perhaps on that account, she had no caresses, nor affectionate words to offer her in return. From the time when she began to complain of a pretty violent pain in the head, attended with vomiting, she insisted on always sitting in her mother's lap, kissing her repeatedly, and addressing her so tenderly, that the poor mother was deeply moved. The disease (for it was incipient cerebral fever) had gone on for three or four days, when the child who was sitting near a window called out, "Oh mamma! how strange! look at that little boy who is running after his hoop in the street; he has only half a blouse and half a face!" This hemiopia lasted a few minutes only; but the child's persistence

and astonishment made such an impression on the mother, that she told us of the circumstance the first time we called.

About ten years ago, I was sent for to see an English boy, 12 years old. He was a very good violinist, and his father, himself an eminent artist, superintended his musical studies. One day, on his playing false, and on his father complaining of it, he answered that the music was badly written, and that he only played what he saw; but as he repeated the same fault several times again, his father took the violin from him and played correctly. The boy, however, asserted that he did not play the music as it was written, and reading it aloud, transposed as he did so, and changed the bars. He used at that time already to complain of headache, and the aberration of sight was the prelude of a cerebral fever which broke out a few days later, and carried him off—as this terrible and inexorable complaint always does.

In the *second stage*, a delusive quiet and rest follow upon the sleeplessness, the febrile action, and the cephalalgia. The child's friends, and even the practitioner, if not on his guard, are deceived by this apparent calm and believe in an improvement which is soon shown to be unreal. An experienced practitioner is too well forewarned by the symptoms of the preceding stage, which have been described to him or have been observed by himself personally, in order to share in those illusive hopes. He is aware that the cerebral fever has entered on its *apyretic stage*, and that it will run a fatal course, in spite of the apparent improvement. The *pulse* in this stage takes on special characters. Generally regular in the first stage of the disease (I say, generally, because it sometimes presents even at this period irregularities which should be taken into account) it now becomes remarkably slow, and excessively irregular and unequal. Whereas, in a child from four to five years old, the pulse normally ranges between 90 and 100 in the minute, and in an infant at the breast, between 100 and 120; it falls to 60, 55, 50, and even lower, in the second stage of cerebral fever.

Somnolence contrasts with the agitation which existed at the beginning; and this apparently calm sleep, following on distressing wakefulness, at first delights the patient's friends, glad of catching at the least ray of hope; but within a short time, on seeing this sleepiness persist, alarm is justly excited. It lasts for two, four, or five days. If attempts be made to rouse the child, he utters a few impatient cries and dozes off immediately again. He is no longer alarmed now by the presence of the practitioner, whose sight he previously disliked. Formerly, he exhibited symptoms of annoyance when his pulse was felt, or at the least thing; but now he is indifferent to all that is done to him. His eyelids may be separated with impunity, so

as to examine the state of his pupils; and if his skin be pinched in order to ascertain the degree of sensibility (which in the first stage is sometimes exalted, as was the case in the little child in St. Bernard ward), he shows but momentary impatience, and immediately again lapses into his former sleepy condition. This, gentlemen, is a sign of the most serious import, which you will scarcely meet with in other diseases.

Another symptom now shows itself which is, *per se*, of considerable significance. The child who in the first stage was exacting, capricious, calling for his mother and driving her away, asking every minute for food or drink, and refusing to have what he has just been asking for so pressingly, as soon as the second stage begins, no longer asks for anything, even when he is most violently agitated, and with the most distressing obstinacy keeps uttering the *hydrocephalic cries*, which I shall presently describe to you. When he is offered drink, he sometimes accepts, but he never shows that he is thirsty by his gestures, or by those movements of the lips and mouth which are so characteristic in infants. He seems to have lost all instinctive sensations. This sort of indifference continues to the end; and even in the third stage, when there is intense thirst, he never asks for drink. If he be at the breast still, his mother must needs press him, separate his lips, and insert the nipple between them: he then sucks with avidity, or refuses entirely.

This symptom is all the more important that in other febrile affections attended with brain symptoms, and which might consequently be confounded with cerebral fever, there is generally very intense thirst, which is manifested in a most striking manner.

In the last stage of cerebral fever the child no longer drinks, even when liquids are poured into his mouth, not only because he has not the sensation of thirst, but probably also because his pharynx and tongue are paralysed, as various other parts of the body are.

In the space of forty-eight hours his face exhibits strange phenomena. He from time to time opens his eyes wide, which shine as they do in individuals that are drunk. His face, which is usually extremely pale, blushes for a minute or two; then he closes his eyes again, and resumes his former aspect. This sort of congestion of the face, which recurs several times in the course of the day, is also of value. It recurs less frequently as the disease progresses. Generally, as he thus opens his eyes, and as his face colours up, the child utters a sharp, plaintive cry, which is perfectly characteristic. This, the *hydrocephalic cry*, was first pointed out by Coindet, and it may recur every hour or half-hour, at variable intervals. Although it is most frequent in infants, it is heard also in the case of adults.

This cry is of such value that I must dwell more on its characters. Most frequently it is single, and loud like the cry of a person frightened by some sudden danger. I do not think that it is due to an acute pain, because a child who is in pain generally utters several cries in succession, and is not consoled in a second. Besides, if the cry be indicative of anguish, the expression of the face is rarely that of suffering.

In the majority of instances the *hydrocephalic cry* is uttered in the second or apyretic stage of the disease, but it is pretty frequently heard at the outset and before the invasion of the complaint even; in other words, it may constitute one of the premonitory symptoms. In some cases, again, it is only uttered in the third stage; as in the case of a little girl whom I saw, at the end of August, 1861, in the department of Maine-et-Loire, with Drs. Despérière (of Saumur) and Duclos (of Tours), and who, during the first two stages of cerebral fever, had not uttered the characteristic cry, while in the third stage her friends were distressed by the violence and frequency of her cries.

A practitioner need not have been very long in practice in order to have met with cases in which the *hydrocephalic cry* is heard from the very beginning, and does not cease, even for five minutes, during four, six, eight, or ten days. In such cases, which are the most dreadful form of the disease, and the most distressing to the friends, the poor little patient never sleeps for a moment, but tosses himself to the right and left, rolling in his bed, and not soothed by caresses nor quieted by threats; and one feels surprised that such a frail organization can resist such a prodigious and incessant agitation. It is a strange circumstance, however, that although the progress of the disease is usually a little more rapid in this form, yet the patient sometimes calms down, and from that time the disease runs the same course as in the simplest forms.

Besides the signs gathered from an observation of the patient's face and the *hydrocephalic cry*, there is another sign to which your attention should be particularly called, namely, *retraction of the abdominal parietes*. The abdomen is excavated, hollowed out like a boat, although not tender on pressure. Although I attach much importance to this symptom, particularly as it helps to distinguish cerebral fever from typhoid fever, in which latter the abdomen is usually prominent, you must bear in mind what I told you at the beginning of this lecture, namely, that retraction of the abdominal parietes is not a pathognomonic symptom.

Another phenomenon which deserves more serious consideration, and which must have struck those of you who looked for it, is *irregularity of the respiration*. It was present in the little girl who was in St. Bernard ward, although it was much less

marked in her case than in a great many others which have come under my observation. At times it was very difficult to follow the movements of the chest, when counting the breathing with a watch in hand. First came a feeble inspiration, before a small expiration, then a deeper inspiration with a more prolonged expiration, and next a weaker respiratory movement, and another still weaker, followed at last by a pause. These four respiratory movements were quickly performed; the chest then remained motionless for three, four, five, or six seconds. This observation was made one day, but on the ensuing days the pause lasted ten, twelve, and even fifteen seconds, instead of only from three to six. In a child of two years old, who was once under my care in the Necker hospital, I noted, watch in hand, intervals of rest lasting from thirty to thirty-five, forty, and even fifty-seven seconds. This irregularity of the respiration occurs independently of the slowness of the circulation which characterizes this second stage, for it continues in the third stage, while the pulse then becomes extremely frequent. You will meet with this singular anomaly in no other complaint; neither in idiopathic convulsions of infants nor in typhoid fever. I am right, therefore, in attaching considerable importance to this symptom, which is of greater value than all others in making a differential diagnosis between *typhoid fever with brain symptoms and cerebro-meningitis.* Thus, in typhoid fever there may be as violent and as localized a headache as in cerebral fever; vomiting may be as obstinate; the ordinary diarrhœa may be replaced by obstinate constipation; the swelling of the spleen, epistaxis, rose-spots, and sudamina may be absent; the abdomen may be boat-shaped instead of being tympanitic; the cerebral macula may be developed, although in a less marked manner, but yet sufficiently to raise a doubt; lastly, the pain in the head may be so acute as to cause the patient to utter cries which may be mistaken for the hydrocephalic cry. But it is in cerebral fever alone that the respiration presents the inequality and irregularity to which I have called your attention. This symptom, which is, so to say, pathognomonic, is all the more important, that the *prognosis* in typhoid fever is considerably different from the prognosis in cerebral fever, in the case of children at least. For you are aware that typhoid fever, even when complicated with brain symptoms, is a much less grave complaint in childhood than in youth and in adult age. The same does not apply to cerebral fever, which is nearly always, not to say invariably, fatal. In the course of my medical career, which has already extended over a long period, I have known two cases only terminate favourably. One of these occurred in my wards in the children's hospital, and I had an opportunity of verifying my diagnosis some time afterwards by a post-mortem

examination. The child got well of his acute disease, which left paralysis behind it, however, but he died of dysentery five months afterwards. Dissection disclosed the most unmistakeable traces of the former cerebral affection. The other case was that of a child whom I saw at Boulogne-près-Paris, in consultation with Dr. Blache.

These two instances are the only ones which I have known, I repeat, in my lengthened career, of this complaint terminating favourably; and when to such exceptional cases so very many others may be opposed which terminate in death, it may well be laid down as a law that this complaint is almost always incurable. This statement will perhaps be regarded as exaggerated, and you have doubtless heard parents say that they had children who had been cured of brain-fever; and perhaps you have even heard practitioners boast of having mastered a disease said to be incurable, while others, as experienced and skilful as themselves, confess that they have always failed. The reason of this is that the former men mistook for cerebro-meningitis typhoid fever complicated with brain-symptoms, which gets well in most cases.

But to return to the description of cerebral fever. The *third stage* is chiefly characterized by the *return of fever*. I have said, that in the beginning fever came on in paroxysms of short duration, recurring three or four times in the course of the twenty-four hours, and that although it was occasionally continuous with frequent remissions, it was never very high. In the second stage the pulse, as we have seen, becomes remarkably slow; but in the third stage it becomes extremely frequent, and goes on increasingly so until death closes the scene.

The stupor grows more and more profound. It was already difficult, in the second stage, to rouse the child, who exhibited impatience by his grunts and cries, but who answered still the questions that were put to him; but in this stage no sign of intelligence can be got from him, and the most powerful irritation can scarcely rouse him. The stupor is much more profound than that of the gravest forms of typhoid fever, for in the latter, there is usually marked agitation co-existing with other signs of ataxy, there is mussitation, carphology, and delirium, sometimes quiet and sometimes noisy. In the third stage of cerebral fever, although the patient's aspect does not, at first sight, very notably differ from that of an individual labouring under typhoid fever, the prostration which exists indicates a much deeper organic lesion of the brain. Delirium is at this period very rare, but it is sometimes present in the first and second stages, although it is very rare even then. Sometimes, but rarely (especially if the child be above four years old), *convulsions* may occur in the first stage of cerebral fever,

but they do not show themselves in the second or apyretic period, or they assume at least a different form, and resemble then epileptic vertigo. The patient opens his eyes suddenly and stares fixedly; but this partial, convulsive movement shows itself more in the third stage simultaneously with symptoms of *paralysis*.

Strabismus is occasionally noted at the onset of cerebral fever, and as it pretty commonly occurs together with convulsions, it may be ascribed to spasm of some of the motor muscles of the eye. But the squint which appears, and pretty frequently continues towards the close of the second stage, and nearly always in the course of the third, is owing to *paralysis*, because there is evident palsy of other muscles supplied by the third or sixth pair. The third nerve is the one most commonly affected; the patient opens one eye less than the other, from the levator palpebræ on that side having lost some power. Strabismus and dilatation of the pupils (which precedes and accompanies the strabismus), and the diminished power in raising the upper lid, are not the only signs of paralysis, for other regions of the body are also affected. Thus, if while the child is lying on his back, the soles of his feet be tickled one after the other, it is found that he withdraws one leg more powerfully than the other. Mobility is, therefore, impaired on one side, and sensibility is affected as well on that side, because the child apparently feels any irritation of the skin there only when it is considerable and prolonged. The persons about him also notice that he has greater difficulty in lifting one arm than the other, and that he lets it drop alongside of his body; on testing it, sensibility in that arm is also found to be diminished.

The paralysis which occurs in cerebral fever presents this remarkable feature, that it seems to shift about from one hour to the other. One day, for instance, the right leg is found to be drawn up with greater energy than the left, when the sole of the right foot is tickled, but on repeating the examination a few days afterwards, you are surprised to find that it is the left leg which now feels and moves better than the right. It would seem from this, as if the paralysis had shifted from one side to the other; but such is not the case: the limb which was first palsied is still so, but the illusion arises from the circumstance that the palsy has not increased in degree in the first limb, while the second limb has become involved to a greater degree. Motor power has not returned in the former, but has been more gravely impaired in the latter. The lesions which are found after death subsequently account for these facts. When the right limbs alone were paralysed, the brain is found to be disorganized on the left side; but when the paralysis apparently shifted from one side to the other, both hemispheres are found

diseased, but more deeply and more extensively on the opposite side to that of the limbs which were most palsied. This apparent mobility of paralytic symptoms more frequently occurs in cerebral fever than in any other complaint.

In this stage there pretty often occurs, as in grave fevers, particularly in typhoid fever, serious inflammation of the eye and ulceration of the cornea, from the absence of winking. Sensibility being either abolished or deeply impaired, and the muscles of the eyelids moving imperfectly only, the lids remain half opened, so that the conjunctiva gets inflamed and becomes the seat of a considerable sanguineous suffusion: the cornea being constantly exposed to the air, and being no longer moistened by the tears, becomes dry, ulcerates, and is at last perforated. This last accident rarely occurs, but there is, in nearly every case, congestion of the conjunctiva and pretty abundant secretion of mucus. Convulsions, which are rare in the first stage of the disease, and in the second assume the form of epileptic vertigo, show themselves again in the third stage, and constitute an important feature of it. They are sometimes inward convulsions, and sometimes consist of regular eclamptic seizures. Thus the child's face is seen to be contorted at times, his eyes roll upwards and inwards, and are the seat of slight oscillations, and his jaw moves as if he were chewing. The thumb is turned into the palm, and the fingers fixed over it; and then perfect relaxation follows on these contractions. These convulsions, which are almost exclusively tonic, sometimes recur for hours together, and affect not only the limbs and face, but the muscles of the larynx also and the diaphragm, impeding respiration to a considerable degree.

In proportion as the complaint draws to a fatal termination, the convulsions become general and assume the form of grave eclamptic seizures. They recur every hour or every half hour, and even oftener, and it is after one of these attacks generally that the child dies in a state of semi-asphyxia. In other cases, death supervenes during profound coma, and trembling of the limbs, subsultus, tendinum, and carphology are the closing symptoms of a more or less prolonged agony.

It very often happens, gentlemen, that an arrest takes place in this fearful development of symptoms, and that the patient who, for several hours or days, was in such a condition that death was thought to be impending, seems suddenly to return to life. He wakes up from his stupor, recognises, or seems to recognise, the persons around him, answers their questions and converses with them; and one must have a sad experience of this complaint in order not to share the hopes which this gleam of improvement excites in a poor mother's heart; and the practitioner must needs have great courage to moderate that joy which

he cannot share, and which, in a few hours, will be replaced by so cruel a grief.

How often, gentlemen, have I been received with cries of joy by happy friends, but how often also have I been compelled to meet such transports with words expressing my gloomy presentiments! Yet I must confess that, at the beginning of my medical career, I could not help entertaining hopes myself, in presence of such an extraordinary improvement.

What shall I say now, gentlemen, as to the *treatment* of a complaint which involves such a fatally grave prognosis? Many remedies have been used against it, and I have tried them myself, but have failed with all of them; and in the two instances of cure which I mentioned to you as being too rare and too exceptional for modifying the general rule, the credit is due to nature and not to art.

Purgatives, calomel in large doses, or in divided doses according to Law's method, iodide of potassium (which Dr. Otterburg states he has used with good effect), large blisters over the shaven scalp, cold affusions, ice constantly applied to the head, have all been tried by me, and always without success.

Next, by instituting a comparison between the results of energetic treatment, and those of the expectant method, I found that death came on at an earlier date in the first class of cases than in the second.

Yet, however convinced I may be of my powerlessness, I cannot decide on remaining perfectly passive, and although taught by a long experience that my efforts will be unsuccessful, I still try to struggle, and by so doing I, at least, do not crush all hope in the patient's friends. I keep up their courage, and do not cause them to regret afterwards that they did not try to save the child. But, convinced as I am also that too active a treatment more promptly exhausts the vital energy, I try to do the least possible harm, since I can do no good.

Calomel, in very small doses, and given more with the view of purging than as an alterative, musk suspended in syrupus ætheris, and antispasmodics are the simple remedies to which I have now recourse when I am free to act. I feed the patient at the same time, and I regard light feeding as the best means of prolonging his life a little more.

When after death the nature of the *anatomical lesions* of cerebral fever is determined, the inexorable gravity of the prognosis becomes intelligible, as well as the powerlessness of the practitioner.

Here is, gentlemen, the brain of the child who gave rise to this lecture. At the base, on a level with and behind the *optic commissure*, the meninges are thickened and infiltrated with a purulent fibro-plastic material. The infiltration does not, in this

case, extend to the fissures between the cerebral lobes, where it usually is very marked; and, as happens in some rare cases, there is no tubercular matter to be seen, either accumulated in masses or scattered here and there, nor are there any grey, transparent granulations, of variable size, but generally not larger than grains of semola.

On making incisions through this brain, we come to the lateral ventricles which contain some rather turbid serosity. The great nervous centres, the fornix, septum lucidum, corpus callosum, and floor of the ventricles are perfectly softened; the cerebral mass is reduced to a pulpy condition.

In the *Lungs*, which you see here, there are no traces of tubercles, nor are there any in the bronchial glands; the mesenteric glands were not tubercular either. On this point, this case is an exception to the rule, for of thirty children, who die of cerebral fever, dissection reveals the presence of tubercular deposits in twenty-nine.

This case seems to me to prove once again that cerebral fever, when said to be idiopathic, that is, occurring in individuals that are not tubercular, does not run a different course from the one it affects in tubercular persons. The prodromata alone differ; one may conceive how the more or less rapid development of granulations and tubercular masses in the meninges gives rise to peculiar symptoms which constitute the prodromic stage of cerebral fever; just as the development of granulations in the peritoneum or the pleura is accompanied by peculiar symptoms. But when acute pleurisy or peritonitis sets in, the presence of these granulations has no influence on the symptoms of the first stage of the disease, and will only influence its termination. It must be added, however, that the presence of granulations and tubercles in the meninges, is such a powerful cause of congestive flux to the brain, that the children must sooner or later die of inflammation.

I reject the name of meningitis for cerebral fever, because the lesions of the meninges seem to me to be secondary only, and much inferior in importance to the deep anatomical alterations seated in the brain itself, such as the softening which destroys the fornix, the septum lucidum, the corpus callosum, thalami optici, and posterior part of the cerebral lobes, to a more or less considerable extent. Hence, if the disease should be named after the organic lesions which characterise it, it ought to be called *cerebro-meningitis*.

Chronic Hydrocephalus.

The cerebro-meningitis of which I have just now spoken, differs greatly from, and is never the starting-point of what is

called *chronic hydrocephalus*, an affection of which you may now see an instance in a young child in St. Bernard ward.

The first thing which strikes an observer, when he looks at an hydrocephalic individual, is the enormous size of the head, out of all proportion with the rest of the body. You have seen the child in the ward: when he was admitted, the circumference of his skull measured 50 centimètres (20 inches) on a level with a line drawn a little above the eyebrows. Cases have been recorded, and I have brought here from the anatomical museum of our faculty, this head, which you may see, and which measures 1 mètre (40 inches) round its circumference. On opening it, it was found to contain within its ventricles, 30 lbs. of fluid and more. Frank mentions a case in which the fluid effused amounted to 50 lbs.; in another case, the circumference of the skull measured 52 inches.

You have observed the peculiar deformity of the head of the child in my wards, and although it is not exaggerated in his case, it still gives you an idea of what it may be in hydrocephalus. There is, first, a considerable disproportion between the face and the skull, the former looking excessively small, for the very reason that the latter is enormously developed, and because also the frontal bones project enormously forwards on a level with the superciliary arches, so that the orbits are pushed down, as it were, and the vertical diameter of the face is, therefore, diminished. This disposition, according to Camper, suffices to enable one to recognise hydrocephalus. Moreover, the two frontal bones separate from one another, from their median suture, which is incompletely united in a child, widening more or less. The same obtains with the sagittal and lambdoidal sutures, the two parietal bones separating from one another and from the occipital bone, which, like themselves, is pushed outwards. The cranial bones are, therefore, soldered together at the base only and fall back (if I may be allowed the comparison) like the petals of an opening flower. On looking at the patient's head, one might think that it was soft, for, when it is moved, undulations are noticed at its upper part, and these are again produced when the child cries or draws in a deep breath. The upper part of the head expands, and is raised during forced expiration, but not during inspiration. By applying the hand over this deformed skull, the separation of the bones may be detected, and this is particularly marked about the fontanelles. The interval between the parietal bones and between the latter and the two frontal bones, may sometimes measure 15, 20, 30 centimètres (6, 8, 12 inches), and even more. Over those parts, the cranial cavity is merely closed by a soft membrane, the pericranium. In some cases, small wormian bones are found in these membranous spaces, in vari-

able numbers. (There is one of these at the posterior part of the sagittal suture, in the child in St. Bernard ward).

Hydrocephalus may last for a long time, especially when it tends towards a cure (a very rare mode of termination). It may be stationary at least for four, five, six, and ten years, as in cases on record, and even more; for individuals who were afflicted with this complaint almost from birth, have been known to live to a very advanced age. Frank relates the history of two men, one 72 years of age, and the other 78, who were hydrocephalic from birth. In such cases, the wormian bones increase in number, and become centres of a process of ossification by which bony causeways are, as it were, formed between one bone and another, indicating a tendency to union, which is always incomplete, however. This enormous enlargement of the skull can only be accomplished, as you may conceive, by distending the skin; hence, after a certain time, from the integuments yielding less easily, the distension takes place at the expense of the contiguous parts, namely of the face, and, especially, at the expense of the skin of the eyelids. The physiognomy of the patient henceforward assumes a peculiar and extraordinary aspect. The eyebrows are pulled upwards, so that the projection of the upper ridge of the orbit which they previously concealed is left exposed; while the upper lid, in consequence of the same traction, becomes too short to cover the eyeball, which seems to project, and to look down and towards the lower lid. In nearly every instance, there is then weakness of sight, or even complete blindness; and, as in congenital blindness, the eyes (which, in hydrocephalus, remain bright and clear) do not gaze at anything, and are the seat of nearly incessant oscillations.

The patient looks sad, but he generally has no pain. Commonly also, the general health seems to be triflingly disturbed; the child, if at the breast, takes it easily, and all his functions are performed regularly. In a certain number of cases, however, hydrocephalus is pretty frequently accompanied, at the outset, by convulsive phenomena. This was the case in the child in St. Bernard ward. When only three weeks old, and therefore almost at birth, he was seized with convulsions, which recurred from four to six, eight, ten, and even twenty times in the course of twenty-four hours. Three months ago, his mother brought him to me for the first time on account of those convulsions, the cause of which I could not make out, for nothing could make me suspect hydrocephalus, as the head was then of normal size. Eclampsia may, therefore, be the only symptom announcing the invasion of the disease, and it is caused by the sub-inflammatory condition of the serous lining of the cerebral ventricles, which condition also brings on the

serous effusion which is poured out into those cavities. The frequent recurrence of convulsions for some length of time should even cause a medical man to suspect the possible supervention of hydrocephalus. In the case of the child, at present in the ward, the convulsive seizures recurred for two months and a half before the head began to enlarge. These convulsions generally increase in violence by degrees, and it very frequently happens that when they have lasted for a certain time, the patient is carried off by an attack of cerebral fever, and the lesions of cerebro-meningitis are then found, on dissection.

When hydrocephalic individuals die of some intercurrent affection, and an opportunity is thus afforded of examining their heads, the ventricles of the brain are found to be enormously enlarged; the brain, the convolutions of which are flattened, is generally sound at the base, but the convolutions of its upper surface are completely effaced and not recognisable from the sulci which have disappeared, and the organ is reduced to a kind of lamina, which the unassisted eye can scarcely recognise as cerebral tissue, the elements of which are, however, made out by the microscope. The membranes themselves (pia-mater, arachnoid, and dura-mater) participate in this thinning, and you may imagine how considerable it must be in those cases in which the fluid effused amounts to 30 and 50 lbs.

Although hydrocephalus almost invariably terminates in death, it may, however, progress very slowly, and I mentioned to you just now, instances of individuals who lived four, five, ten, and even (as in cases recorded by Frank) seventy-two and seventy-eight years. Apart from these exceptional cases, which are not, however, very rare, this complaint lasts habitually a year or two, unless it presented acute symptoms from the beginning, in which case, death sets in rapidly. But how lamentable the life to which the unhappy individuals, whose existence is prolonged, are condemned! What a sad spectacle to those around them! and what a source of continual affliction they are to their parents! So long as they are infants in arms, they can scarcely bear the weight of their head: by and by, when they begin to walk (and they always walk later than other children), their gait is vacillating, and as the disease progresses, they can no longer stand, and are confined to their beds.

Several reasons concur in causing this inability to stand or to sit up. There is, on the one hand, the enormous weight of the head, which is no longer balanced on the trunk; and on the other, blindness, which accompanies hydrocephalus, and which, by rendering the patient incapable of guiding himself, prevents his walking; and lastly, there is a *kind* of paralysis, resulting from the compression of the nervous centre. I say a

kind of paralysis, because the paralysis is not carried to the degree which might have been, perhaps, expected. In the case of the child at present in our wards, although the amount of fluid poured out into the ventricles must be considerable, if we estimate it by the size of the head, there is no symptom of paralysis; the child moves his legs and arms easily, and his bladder freely expels the urine. The reason of this is, that the skull has yielded to the pressure from within, and, as a consequence of its enlargement, the brain has escaped compression. But when hydrocephalus has reached such a degree that the skull can no longer expand, there comes a time when compression is unavoidable, and the functions of the organ being abolished, loss of motility results.

Even when the disease is stationary for a very long period, there is arrest of mental development; the intellect fails, and this failure generally passes into nearly complete imbecility.

In all these cases, therefore, prognosis is of the most serious character, and medicine is always powerless to cure or even to relieve the sufferer. Yet there has been no lack of methods of treatment. With the view of combating the sub-acute inflammation which causes the effusion, purgatives, calomel, and even blood-letting, have been recommended; diuretics, sudorifics, and sialagogues have been vaunted.

Methodical compression of the head has been particularly lauded, and I long had recourse to it myself; but I have now completely set it aside, on account of a case which fell under my notice.

I was once consulted about a child, five months old, who was suffering from chronic hydrocephalus, and whose head was of about the same size as that of the child in the ward. I had hopes that, by compressing the head by means of strips of sticking plaster, I might prevent a further increase of the effusion: at the end of a week, I went to see the child, and to apply fresh strips of plaster, after removing the old ones. The size of the head had appreciably diminished, but the child died suddenly, five or six weeks after the second application of the compressing plaster. He had suddenly cried out as he was going to take the breast, a copious flow of liquid had taken place through the nostrils, and the head had shrunk like a bladder which empties itself. Now, what had occurred? As the compression of the upper part of the cranium prevented a further effusion of liquid, the base of the skull had yielded, as it does when, in order to separate the cranial bones, anatomists fill it with water and haricot beans, which latter, on swelling, disarticulate the bones. In the case of my young patient, the base of the skull had yielded to the pressure of the fluid, disarticulation had occurred, and the fluid, finding

a channel through the æthmoid bone, had flowed out through the nasal fossæ. Death had then resulted from the sudden change which had taken place in the anatomical conditions of the brain.

The brain has been tapped through the sutures and fontanelles by celebrated surgeons, and the operation has been even repeated several times on the same individual; but many of those who had praised it at first have finally proscribed it, for its advantages do not counterbalance its disadvantages. Of late, iodide of potassium has been very much lauded; and for the last few years, I have myself used iodine lotions to the head. I give iodide of potassium internally at the same time, in doses of two grains at first, which I gradually increase to four, five, six, and even eight grains, according as it is tolerated. The end which I have in view by prescribing iodine lotions is to favour the absorption of the effused fluid, guiding myself on the success obtained by means of these lotions, in effusions into the serous membranes of the pleura, the abdomen, or the joints.

LECTURE XVII.

ON NEURALGIA.

Neuralgia is generally a symptom either of a local lesion, or, more commonly, of a general affection. — Cutaneous hyperæsthesia over the peripheral expansion of the nerves, followed sometimes by anæsthesia.— The painful spots indicated by Valleix are not accurate.—The spinous processes of the vertebræ are always tender on pressure, but the fact was not mentioned by Valleix.—The cause of a neuralgia influences its seat. — Periodicity and intermittence are frequent characters of neuralgia, whatever be its origin.

Gentlemen,—I am averse to treating in this place questions relating to pathology, as they should be discussed elsewhere, but when several cases of the same disease occur at the same time in our clinical wards, or when a remarkable case, full of interest, comes under our notice, it is my duty to take the opportunity of pointing out to you how far clinical cases differ from, or resemble, those which are generally regarded as types; for this practical study, based on observation, leaves in your mind recollections which cannot be effaced, and prepares you, in a remarkable manner, for a study of pathology, which can never be entered upon and completed unless controlled by clinical observation.

Strangely enough, we have at present in our wards four somewhat remarkable cases of neuralgia. At No. 7, in St. Bernard ward, there is a woman who is suffering from hepatic and intercostal neuralgia, following hepatic colic; at No. 12 is another woman labouring under rheumatic neuralgia; and at No. 13, a third suffering from neuralgia of nearly all the branches of the lumbar plexus, following on a sub-aponeurotic abscess of the iliac fossa; and lastly, at No. 23, there is a chlorotic girl afflicted with neuralgic pain in various regions, as so frequently occurs in chlorosis.

For the last two or three months you have also had occasion to see other cases of the same kind, to which I called your attention at the time, so that I can now point out to you in a few words the chief forms of neuralgia, and the various modes of treatment to which I have recourse. Let me first remind you that the majority of pathologists have divided neuralgias into two great classes; namely, those which are not due to an organic lesion, of which they are a sympathetic expression, and secondly,

those which are dependent on a more or less grave lesion, involving some nerve branches, or compressing or irritating them.

I do not wish to find fault too much with this division, which may aid memory, and may help one to understand neuralgias better; but I wish to observe that, after all, all neuralgias are only symptomatic. Surely there are notable differences, which I shall presently point out, whether the neuralgia occurs, as it so frequently does, in chlorosis, or in chronic lead-poisoning, or in anæmia from various causes, and in rheumatism, or in cases of carious teeth, of necrosed bone, or of a tumour in the pelvis, or of phlegmonous inflammation of that cavity; but whether the neuralgia be due to chlorosis or to a carious tooth, it is still a symptom, in the first case, of chlorotic cachexia, in the second, of the caries of a tooth. As we shall see presently, there is a great difference between these two forms of neuralgia, as regards their obstinacy and their degree of curability, but not as regards *pain*. All neuralgias, regarded as painful affections, resemble one another, with the exception, however, of that neuralgia which I have called *epileptiform*. It is certainly true that the cause of the neuralgia most frequently possesses a manifest influence on the recurrence, the duration, and the period of invasion of the paroxysms of pain as well as on the seat of the pain, but the pain itself exhibits very nearly identical characters. If you recall to mind how I looked for and found painful spots, you must be convinced that the form of the pain did not vary, whether chlorosis, syphilis, rheumatism, marsh miasmata, or an acute inflammatory or a chronic affection, had caused the neuralgia. When the branches of the fifth cranial nerve were affected, the most painful points were at the exit of the ophthalmic of the superior and of the inferior maxillary branches; next to these came the frontal point, which was rarely absent, and then the parietal point, which was less frequently met with; lastly, the occipital nerve was nearly always simultaneously affected, although its origin is independent of that of the trigeminal. It is a strange and inexplicable fact, but which has been constantly present in all the cases which we have carefully observed and noted, whether the fifth was affected by itself, or the occipital nerve as well; pressure made on the spinous processes of the first two cervical vertebræ *always* caused pain, and, in a certain proportion of cases, immediately brought on shooting pain in the diseased nerves.

When the nerves of the brachial plexus were affected, pressure made over the spinous processes of the last cervical vertebræ gave pain; and the same thing occurred when the spinal column was examined in cases of intercostal, lumbar, or sciatic neuralgia.

It may, therefore, be stated in general terms that, in neuralgia, the spinous processes of the vertebræ are tender on pressure at a spot nearly corresponding to the point of exit of the nerve from the intervertebral foramen, and that the pain pretty frequently extends a little further up along the vertebral column. Thus you have seen, how, in individuals suffering from sciatica, pressure on the sacrum gave pain; while, in females afflicted with neuralgia of the lumbar plexus, there was tenderness on pressure of the last dorsal vertebræ.

It might seemingly be inferred from this fact, that the starting point of the neuralgia is perhaps in the spinal cord, and that the peripheral is only an irradiation of the spinal pain. Yet I confess that it may be equally admitted that the lesion of the periphery of the nerve trunk, or of some portion of it, transmits to the cord the painful impression which is made so acute by pressure on the spinous processes. Let me add, that the latter view is the more probable of the two, since, in most cases, marked peripheral lesions are the starting point of neuralgias, as in cases of decayed teeth, of necrosed bones, of tumours of various kinds developed either in the vicinity or in the substance of nerve trunks, or of inflammations including nerves within their area. On the other hand, it cannot be denied that frequently, particularly in rheumatic affections of the spinal cord, the disease begins in the nervous centre and radiates from it to the periphery.

Whatever be the explanation, however, neuralgia reveals itself by acute pain, when pressure is made on the spinous processes which correspond to the origin, or the point of exit, of the diseased nerves. I have told you, that this applied to the fifth nerve, and that, in no case which I had examined, had I failed to find great tenderness on pressure of the first two cervical vertebræ, as well as of the trunk and branches of the nerve. It is true that in such cases, although the phenomenon is constantly present, it is not so easily or so satisfactorily explained. In neuralgia of the lumbar plexus I can pretty easily understand, from the anatomical condition of the parts, how pain may be excited by making pressure on the spinous processes of the last dorsal, and the upper lumbar vertebræ, but I do not see what relations exist between the first two cervical vertebræ and the trigeminal nerve.

As this phenomenon is almost invariably present, it becomes a valuable element for diagnosis. When, after some external violence, an individual complains of a stitch in the side, there is no tenderness of his spinous processes, and this is also the case at the onset of an attack of pleurisy or of pleuro-pneumonia. Neuralgia is not yet developed; there is merely local pain, which sometimes, however, passes into neuralgia at a

later period. But if pain in the side sets in in an anæmic, or chlorotic, or dyspeptic patient, of course, apart from all local lesion, there always is tenderness on pressure of the spinous processes of the vertebræ.

I will give you another illustration. In toothache, arising from the presence of a false tooth with a pivot, the spinous processes are not tender on pressure, however acute the pain may be; but if this pain, which is at first limited to the locality of the tooth, in the lower jaw, for instance, extends to the inferior maxillary division of the fifth, then to the superior maxillary branch, and lastly to the ophthalmic, the spinous processes then become tender on pressure, and the case is one of neuralgia.

The same thing obtains in hepatic colic. Fearful pain sets up suddenly in the pit of the stomach, and in the region of the gall-bladder, and of the ductus communis choledochus. So far there is merely local pain, without neuralgia, and there is no tenderness on pressure of the dorsal spinous processes; but after two or three days spent in acute pain, a sharp pain is frequently complained of in the seventh, eighth, and ninth intercostal spaces, in the shoulder, in the neck, and in the arm on the same side; from that time, neuralgia exists, and the vertebræ become very tender on pressure.

You see, gentlemen, that the apparently subtle distinction which I established just now is founded, and that local pains should not be mistaken for neuralgias, as we possess a precious sign, which enables us to distinguish them. It seems also, in the cases which I have just cited, and in which a local pain gives rise to neuralgia, that the spinal cord is influenced, and then, through reflex action, excites neuralgia, in which it appears to be always involved.

The general condition of the individual, cachetic states in particular, plays an important part in the development of neuralgias. If we find, that persons suffering from chlorosis or from a rheumatic diathesis have a strange liability to neuralgia, which, as it were, developes itself spontaneously in them, it is conceivable that, in such individuals, an acute pain or a very painful tumour will give rise to irritation of the spinal cord, from the centre of which neuralgias will develope with extreme violence. This is, indeed, what occurs in such cases. Whereas, for example, in a woman of robust constitution, chronic inflammation of the uterus or its appendages may exist for a long period without exciting neuralgia, the least irritation of those same parts will, in a chlorotic female, bring on neuralgia of the thighs, of the groin, &c. We had an illustration of this in the case of a young girl who occupies bed No. 27, and whose history I will relate briefly to you. "This young girl is

seventeen years old; she has menstruated regularly until this last time, when on her taking a cold bath on the last day of her menstrual period, the menses were immediately suppressed, and she shortly afterwards felt an acute pain in the region of the left ovary. Within a few days she had palpitation of the heart, got out of breath easily, and complained of disordered digestion and of vague pains; she had become chlorotic. She was then admitted under my care, on account of an acute pain in the chest, which she complained of. The pain was so intense that it impeded respiration, but I easily made out that it was merely due to intercostal neuralgia on the left side. As this was apparently dependent on a chlorotic condition, which generally gives rise to neuralgic pain in various regions at the same time, I looked out for some other neuralgia and discovered a lumbo-abdominal and a crural neuralgia, both on the left side also. The patient did not complain of them, however, as her attention was wholly engaged with her intercostal pain which partially interfered with her breathing."

The many examples which I have related to you, and your personal examinations of the spinal column of individuals suffering from neuralgia, have, therefore, shown you how tender on pressure the spinous processes are over the spots corresponding to the presumed lesion of the spinal cord.

There is another peculiarity to which I have drawn your attention at the bedside, and on which authors, who have written on neuralgias, have not been explicit enough, namely, *cutaneous hyperæsthesia* at the points of exit of the nerve-trunks. This phenomenon is best studied in cases of intercostal, lumbar, and crural neuralgia. Its characters are such that they cannot be mistaken, and may almost be regarded as invariable. When the skin is scratched with the tip of a nail, or is gently rubbed with a hard body, as the blunt end of a pencil, the patient complains of a pricking pain, of a sensation of burning, which he compares to that felt on rubbing a portion of skin which has been burnt to the first degree. The acuteness of the sensation varies according to individual conditions difficult to appreciate, for it is somewhat dull in some and singularly exalted in others. In those regions where the nerve-trunk, from being deep-seated, becomes superficial, as in the case of the external popliteal, and the internal saphenous nerves, the track of the painful nerve may be followed with the tip of the finger as far as its cutaneous expansion. In the case of the intercostal nerves, which at their exit break up immediately into extremely numerous branches, the cutaneous pain spreads over a somewhat considerable area, instead of being circumscribed as in the above instances. This circumstance gives rise to errors in diagnosis every day, and it must be

admitted that the patients themselves greatly contribute to mislead us. You remember the young woman who lay at No. 10, in St. Bernard ward. She had several very mobile neuralgias, and you very often heard her complain of an acute pain in the stomach, which I tried to calm by the internal administration of bismuth, belladonna, opium, &c. One day, on looking out very carefully for the tender spots of an acute intercostal neuralgia of which she complained, I found exquisite tenderness of the spinous processes of the sixth and seventh dorsal vertebræ, and extreme exaltation of cutaneous sensibility over the parts to which was distributed the anterior extremity of the two affected intercostal nerves; and as these nerves send terminal branches to the whole of the epigastric region, the skin over it was extraordinarily painful. I easily understood, then, that the case was not one of *gastralgia* but of *epigastralgia*, and it gave me the key to the interpretation of many cases which had formerly seemed very difficult to me. When once my attention was drawn to this point in pathology, I saw a good many analogous instances, both in private and in hospital practice.

It is common, indeed, to meet with women who cannot bear a string round their waist, or the least pressure over the pit of the stomach, and, on examining them, it is found that in nearly every case there is intercostal neuralgia with epigastralgia. It is very common also to meet with women who complain of cardiac pains which are merely due to intercostal neuralgia, and as this neuralgia is peculiarly frequent in chlorotic and nervous women, that are more than others liable to palpitations of the heart, it follows that both the patient and her medical attendant believe that there exists a connection between the palpitations and the pain in the side, while the connection is merely apparent. You saw very recently a curious instance of this in a young girl who occupied bed No. 9, in St. Bernard ward, during the month of November, 1863. She complained one morning of violent palpitations of the heart, and at the same time of acute pain over the cardiac region. It was easy to see that her heart beat more quickly and more strongly than usual, and a soft blowing murmur was audible at the base, coinciding with a continuous blowing murmur in the vessels of the neck; the patient was chlorotic. You remember that, on examining the cardiac region, as I generally do, to look out for intercostal neuralgia, I found considerable hyperæsthesia of the skin, together with tenderness on pressure of the spinous processes of the fourth and fifth dorsal vertebræ. She besides complained of neuralgic pains in various other regions, in the face, in other parts of the trunk, and along the lower extremities. These cases, to which I might add a good many more, prove to you sufficiently the importance of this

hyperæsthesia as a symptom, and how it may give rise to many errors when it is wrongly interpreted.

But, gentlemen, there is another phenomenon, quite the reverse of the above, which sometimes, although more rarely, exists in neuralgia, namely, *anæsthesia*. It often follows on idiopathic non-spinal neuralgias, that is, on neuralgias apparently of rheumatic origin, or due to a slight lesion of the cord. At the outset, and often for a somewhat lengthened period, there is only an exaltation of sensibility; but when the affection has lasted a long time, the exaltation is replaced by a diminution, and lastly by a complete loss, of sensibility. In such cases, I admit, there is something more than a neuralgia, and the anæsthesia may be regarded as the consequence of a change in the structure of the cord or of the nerve trunk, as occurs in cases of neuritis. It is still pretty frequent to find cutaneous anæsthesia succeed hyperæsthesia, especially in herpes zoster. This complaint, as you are aware, is often attended with persistent neuralgic pains, and I have known these to last sometimes more than ten years, until the patient's death. When the pains have lasted for a long time, however, it sometimes happens that the hyperæsthesia is followed by a curious kind of insensibility, of which the patient complains bitterly. Anæsthesia comes on pretty frequently also in the course of sciatic neuralgia, particularly over the area of distribution of the branches of the external popliteal nerve, but only when the affection has lasted a very long time.

I have always felt surprised, gentlemen, at the facility with which the profession accepted certain views respecting the diagnosis of neuralgia propounded by Valleix,[1] namely, the *superficial tender spots*. You have witnessed the scrupulous care with which I look for these spots in every one of my cases. In cranial neuralgias the spots are those indicated by Valleix, and they were already known before him. The fifth pair divides into three principal branches, and it is at their point of exit that pain is most particularly felt, namely, over the supra-orbital notch, where the ophthalmic branch becomes superficial, over the infra-orbital foramen which gives passage to the superior maxillary branch, and over the mental foramen, through which emerges the inferior maxillary division of the nerve. This is easily ascertained by making pressure over those spots with the blunt extremity of a pencil, and even with a finger. But there are other tender spots as well. When the supra-orbital neuralgia is rather intense, the nasal branch is extremely tender, and pain is excited by gently compressing the skin over the point of exit of this small nerve. Although

[1] Traité des névralgies, Paris, 1841.

the ophthalmic branch, on leaving the supra-orbital notch, breaks up into numerous ramifications over the forehead, and although on dissection no branch can be seen, like the nasal, becoming suddenly superficial from being deep-seated, yet pressure generally causes a somewhat acute pain over the frontal eminence. Pressure also over the parietal eminence is slightly painful, although that branch of the fifth which goes upwards in front of the ear, and ramifies in the scalp as far as the parietal eminence, is not subcutaneous like the nasal branch of the ophthalmic. There is another remarkably tender point in the track of this small branch, namely, over the zygomatic process in front of the ear.

After all, the fact observed by me is in accordance with others already made out; for when nerves are affected with neuralgia, there are two points where pain is most felt. 1st, where the branch, after being deep-seated, becomes superficial; 2nd, where the branches and small twigs of the nerve terminate in the skin. There is nothing surprising, therefore, in the fact that the small temporal branch and the ophthalmic division of the fifth are painful only in the two extreme points which I have just indicated.

As to the occipital branch, it is generally painful where it comes out of the substance of the muscles, and it is tender for a pretty short distance.

Valleix was guided by his anatomical knowledge more than by facts, when he pointed out the spots where pain was particularly felt. Seeing, for instance, that in the face pain was chiefly felt over the points of exit of the nerves, namely, where they issue from the bones, and become subcutaneous, he thought that the same thing occurred in the case of other nerves, but this is not borne out by observation. He then indicated three tender spots in intercostal neuralgia, which is such a common affection; the first, situated over the angle, the second, about the middle, of the rib; and the third, about its sternal end. Now, his assertion is perfectly unfounded, and a few days' researches are sufficient to prove it.

But where, after all, are these tender spots found? They are three in number, of which two are more important than the third, namely, one which has not been mentioned by Valleix, but is of considerable value in diagnosis, and which I have termed the *spinous point*; and another, which is nearly as important, which I have called the *spot of peripheral expansion*. The *spinous point*, as its name indicates, is situated over the spinous processes of the vertebræ, and since my attention has been drawn to it, I have never known it to be absent. It is easily found out by making pressure in succession on the spinous processes of the vertebræ, beginning with the first

two, immediately beneath the occipital bone, down to the loins. When the tender spot is reached, the patient makes an abrupt movement, and tries to avoid being touched, and sometimes even cries out. Pressure on the vertebræ above and below the point gives no pain.

The *spinous point* is met with not only in neuralgia of the face and of the trunk, but also in sciatic neuralgia, as was the case in two women suffering from this affection, who were under my care in November, 1863. Before I examined them, I must say that I expected to find tenderness on pressure of the spinous processes corresponding to the lumbar swelling analogous to what is met with in intercostal neuralgia; but to my great surprise, no pain was produced by pressure, however strong, made on the spinous processes. Pressure on the sacrum, however, gave rise to the same kind of pain as that felt on pressing the dorsal vertebræ in intercostal neuralgia. This circumstance is probably owing to the neuralgia only beginning on a level with the sacral plexus.

In one of these cases, the neuralgia was of an erratic character, and you could observe the great difference which exists, in this respect, between sciatic neuralgia and neuralgia of the crural nerve and of other branches of the lumbar plexus. For the sacrum ceased to be tender on pressure as soon as the neuralgia disappeared, and deep pressure made over the spinous processes of all the lumbar vertebræ gave no pain, while pain was complained of when the eleventh dorsal vertebra was pressed, which nearly corresponds to the origin of the nerves of the lumbar plexus.

You may perhaps recollect a woman who lay in bed No. 14, in the nurses' ward, who suffered from various neuralgias of a rheumatic origin, and whose spinous processes were tender in several places. When pressure was made on her occipital protuberance, which is the analogue of the spinous processes of the vertebral column, and which may be regarded as the spinous process of the great cranial vertebra, considerable pain was excited in the branches of the fifth, especially in the ophthalmic branch of Willis. In some cases, and by a similar process, a pretty sharp pain is excited in the sciatic nerve by making pressure on the spinous processes of the sacrum. When the spinous point has been detected, the *spot of peripheral expansion* has to be determined; and in so doing, one must be very careful to keep free from error. Suppose the case, for example, to be one of neuralgia of the sixth intercostal space. The spinal point will correspond to the fifth and sixth spinous processes, and the peripheral tenderness will be complained of over an antero-lateral portion of the chest corresponding to the sixth intercostal space. Now, whereas the upper ribs form

nearly a right angle with the vertebral column, the middle and lower ribs form an acute angle, so that these ribs go from below upwards as far as their corresponding cartilages, which are, on the contrary, placed horizontally or even obliquely from below upwards. If therefore, on going from the tender spinous process, one does not keep to the rib and the intercostal space, the relation cannot be made out which exists between the *spinous tender point* and the *spot of peripheral expansion*. For an imaginary line, drawn round the chest perpendicularly to the vertebral column, does not follow the direction of the ribs in order to reach the median line. Now, as the fifth and sixth spinous processes are exactly opposite the middle of the sternum, this is where the tender spot of peripheral expansion is looked for; whereas it is situated three or four inches lower down, that is to say, about the end of the rib, near where the rib joins the sternal cartilage. I have dwelt on this point, gentlemen, because I have, on several occasions, when going round the wards, seen students unable to find out the antero-lateral tender spot in an intercostal neuralgia, after they had succeeded in detecting the *spinous point*. I have told you already, that there is cutaneous hyperæsthesia over the area of peripheral expansion, and how to recognise it. This phenomenon is nearly constant.

When the neuralgia is due to a cachexia, the nature of the latter has also a somewhat marked influence on the seat of the neuralgia. Thus, chlorosis gives rise to neuralgia in several regions at the same time, although the trigeminal nerve and the nerves of the solar plexus are those which are most commonly affected. Gastric and intestinal neuralgia is rarely absent in women whose constitution is enfeebled by uterine hæmorrhages or by leucorrhœa. In the cachexia due to marsh miasmata, the ophthalmic nerve is the one most generally affected. In rheumatic individuals, the occipital and the sciatic nerves are most frequently attacked. These statements must be taken generally, of course, and are liable to very numerous exceptions.

In cancerous and syphilitic cachexias, neuralgia has no favourite seat, but sets up within the area of irritation or of pain developed round some local lesion; thus we meet with sciatic neuralgia in cancer of the pelvis, and with lumbo-abdominal and crural neuralgias in cancer of the kidneys and of the uterus; and with temporal and occipital neuralgias, and brachial neuralgias, when a syphilitic tumour developes itself on the parietal bones or over the humerus.

It is rather strange, gentlemen, that diseases of the nervous system should very often give rise to intermittent phenomena. Epilepsy, catalepsy, certain kinds of chorea, and many other

convulsive affections, pretty frequently assume not only an intermittent but also a periodic type. The same thing occurs with many neuralgias, visceral or external. This *intermittent* and periodic character shows itself even in organic affections of the greatest gravity. Thus I saw a lady, in the year 1845, in consultation with Messrs. Récamier and Maisonneuve, who was affected with cancer of the inner wall of the uterus. She had every day paroxysms of awful pain, recurring at exactly the same time; the pain was seated in the hypogastrium and radiated to the kidneys, to the buttocks, and to the thighs, along the track of the principal nerve-trunks. It lasted from three to four or five hours, and then ceased, to reappear on the following day exactly at the same hour.

In the year 1850, I saw another lady afflicted with the same complaint, with my excellent friend Dr. Lasègue: the cancer involved the inner wall of the womb, and it seemed as if this form of the disease, which is somewhat rare, had the sad privilege of giving rise to intermittent and periodic pain. I have never known another case in which such excruciating pain was felt. When the pain was at its maximum, the unfortunate patient rolled on the floor, uttering fearful groans. It recurred every day, but, strangely enough, it returned from half an hour to three-quarters of an hour or an hour later each time, so that, in the space of a month or more, the hour of attack had gone round the clock. The neuralgic paroxysm did not last more than four or five hours, and then quiet was restored; all pain ceased, and there merely remained a very slight sense of fatigue and weight in the hypogastric region.

Again, in 1862, I saw in Paris, with Messrs. Nélaton and Bouillaud, a Greek lady, who had exceedingly profuse menorrhagia and awful neuralgic pain in all the branches of the lumbar plexus. She had fibrous tumours of the walls of the uterus, protruding into the cavity of the viscus. I attended her for nearly a year, and during that period the pain recurred every day with hopeless violence and obstinacy, between twelve and two o'clock, and ceased about five or six in the day. Sometimes, though rarely, it occurred during the night, but it then lasted a short time only. The patient was nearly well in the intervals, with the exception that she felt extremely weak in consequence of the loss of blood, and of the nervous shock caused by such intense pain. I need not add that, in these three cases, bark in all its forms was largely administered, but that it never succeeded in modifying, in the least, the pain or its periodic recurrence.

You may have seen at No. 32, a young woman whose case I have often quoted, for it is peculiarly interesting, and affords a sad illustration of the influence which a local and persistent

pain may exert on the whole system. She lost her left eye after a grave attack of ophthalmia, but as she preserved a very mobile stump, she has been able to wear an artificial eye, which is moved by the stump, so that it is very difficult to find out her deformity. The artificial eye, however, being a foreign body, has irritated what remained of the globe of the eye, and given rise to a supra- and an infra-orbital neuralgia, just as might have been caused by a foreign body introduced into a carious tooth. For a very long period the affection was limited to the two upper divisions of the fifth nerve, but the continuous pain and loss of sleep have brought on a strange nervous susceptibility, and, subsequently, multiple neuralgias, at first in the other side of the face, and next in the cervical, the intercostal, and the lumbar nerves, etc. You will often meet with the same phenomena, in women particularly, as a consequence of local lesions which have given rise to neuralgia. Thus, it is not uncommon to find a false tooth with a pivot, which at first merely caused a very sharp local pain, and later, neuralgia of that portion of the trifacial nerve which supplies the diseased jaw, ultimately bring on neuralgia of all the branches of that nerve, and at last, nearly general neuralgic pain.

The evolution of the neuralgia commonly takes place after the following manner: when the nerve has several branches, the pain sets up in all the branches in succession, as if the lesion had extended from one branch to the trunk, and thence to the other branches. In the case of a plexus, the connexion between its various branches produces the same effects as those observed in a nerve with a single trunk. This occurred in a woman of 38 years old, who was at No. 13 in the nurses' ward. She had been delivered four months previously, and complained of a lancinating pain in the whole of the anterior aspect of the thigh. The hypogastrium was tender on pressure along the track of the crural nerve, and in the right iliac fossa, but there was no engorgement of the fossa. The pains recurred chiefly about seven in the evening, and lasted two hours; they came on after a frontal headache, which disappeared completely. At the end of six days, a tumour with a round contour was detected in the iliac fossa; five days later there was very marked fluctuation, and the pain in the thigh had increased considerably, and extended into the knee, impeding the movements of the limb. Pinching of the skin on the anterior and inner aspect of the thigh gave great pain. I requested Mr. Jobert to open the swelling, and by means of an incision parallel to Poupart's ligament, this skilful surgeon opened the abscess, which was situated deeply under the fascia iliaca, without wounding the peritoneum, and gave issue to a tumblerful of laudable pus, which had no fœtid smell. The pain in the thigh ceased im-

mediately after the operation, and from that time, the crural and the lumbo-abdominal neuralgia has never appeared again.

In this case, then, the pain was so connected with the existence of the inflammatory lesion that it began and ended with it; and on the other hand, the neuralgic pain began in the branches of the lumbar plexus involved in the inflamed tissues, and extended next to all the branches of the nerve trunk, and, lastly, to all the branches of the plexus.

The connexion between all the branches of a plexus exists likewise between all the nerves that issue from the spinal cord, which may be regarded, to a certain extent, as a real plexus. What I have said respecting the tenderness, on pressure, of the spinous processes, which is so constant in neuralgias of the head, trunk, and limbs, accounts in some measure for the influence which the neuralgia may subsequently exercise on the development of other neuralgias in regions that are very distant from the primary one, although this influence cannot be understood at first sight.

§ 2.—*Neuralgias of rheumatic origin.—Their multiple manifestations.—They frequently alternate with the articular pains.*

At No. 31, in St. Bernard ward, is now a woman who is one of our hospital nurses. This is the fifth or sixth time that she comes under my care. The prominent point in her case is the rheumatic diathesis under which she labours, and which is characterised by the diversity of its manifestations. She sometimes has rheumatism of the spinal cord, with nearly complete paraplegia; sometimes her brain is affected, and she lies in a kind of stupor, without expression in her eyes, while her ideas are confused, her head feels heavy, and she is unable to move. This time, the patient suffered from pain in her joints, both her hands were affected, especially the metacarpo-phalangeal articulations, which were swollen, red, and painful, and then other joints were attacked in succession. A few days afterwards, the articulations got well, and then neuralgic pain set up in various branches of the brachial plexuses, either in several simultaneously, or in succession. A fortnight had scarcely elapsed when the brain became affected again, and the previous symptoms returned, while, after a few days, symptoms of congestion of the cord showed themselves, attended with great feebleness of the lower limbs, a true incomplete paraphlegia. These phenomena disappeared and were replaced by neuralgia of the lower limbs; and at last, about October 20, six weeks after her admission, fresh pains came on in the joints again, proving the rheumatic origin of all the other symptoms. Both knees were swollen and painful, and fluctuated markedly, while at the same time, the external saphenous nerve on the left side was affected with neuralgia.

A week after this the patient felt very little pain in her knees, and had no neuralgia of the left saphenous nerve, but other nerves were attacked: the supra-orbital, the parietal, the occipital, and the frontal, on the left side, and, as is usually the case, there was tenderness on pressure of the spinous processes of the first two cervical vertebræ. At the end of another week, she had neuralgia of the sixth intercostal space on the left side, and exquisite sensibility of the skin over the epigastrium on the same side.

Thus in the space of two months, this patient has presented symptoms of rheumatism, of a flying, but of a very painful nature, affecting sometimes the cerebrospinal axis or its envelopes, sometimes the articulations, and sometimes, again, various nerve trunks. The implication of the joints is proof sufficient of the rheumatic nature of all the phenomena. There is a peculiarity also to which I must draw your attention in this case, namely, that, however various the neuralgias were, they always showed themselves on the left side exclusively.

This case then exhibits the influence of rheumatism on the production of neuralgia; and it frequently happens that individuals suffering from pain, evidently seated in the joints, and attended with swelling, are seized with neuralgias when the joints are free from pain, while they again complain of articular pain when the neuralgia disappears. In some cases, as in that of the young girl who is now at No. 9, and in whom, by dressing with morphia blisters made by ammonia, neuralgia of the trifacial nerve was removed, there may be at the same time pain along the nerve tracks, and pain evidently confined to joints.

I must explain myself further, in order not to leave on your mind erroneous impressions, and to remove as much as possible all confusion arising from the too elastic appellation of rheumatism.

Acute articular rheumatism is a name generally given to a pyrexia characterised by very sharp febrile action, by considerable and generally transitory effusion into the joints, and by a tendency to implicate extra-articular synovial membranes, the serous membranes of the chest and of the brain, and the lining membrane of the heart.

I have not here to justify and defend the name given to this disease, but it differs essentially from another complaint which also attacks the joints, and is known under the name of *gout*. It is as different again from another affection, which is termed *rheumatism*, and which affects joints, muscles, and limbs in their continuity, without nearly ever giving rise to effusions into joints, and in which serous membranes and the heart are never implicated. This last form is certainly the most frequent of all, and is the one which particularly attacks nerve trunks, and

causes those neuralgias, which, from want of a better term, I have called rheumatic. In gout, properly so called, neuralgia occurs occasionally also, but much less frequently than in the non-febrile form of rheumatism. Rheumatic neuralgia is much more mobile than neuralgias dependent on a cachectic condition, as chlorosis, for example. It has a remarkable tendency to attack the brain and spinal cord, but in general temporarily only. In some cases, however, it fixes itself on the nervous centres, and causes paralytic symptoms, probably due to some superficial lesions of the nerve-trunks or their roots.

§ 3.—*Syphilitic neuralgias should not be confounded with the pains due to exostoses.—There is no tenderness on pressure of the spinous processes in cases of pain due to exostoses, or in pleuritic stitch.*

The pains which in syphilis are felt in the bones, simulate neuralgia; and it sometimes happens, as I shall tell you presently, that when the bones are diseased in syphilis, real neuralgia is set up from compression of nerve trunks. Thus, a bony tumour in the pelvis, or an exostosis developed on the track of some branches of the fifth nerve, may give rise to neuralgic pain of extreme violence. When the pain is exactly limited to the seat of the exostosis it no more deserves the name of neuralgia than does the pain caused by a whitlow or an abscess. Yet, I am aware that when the growth is situated exactly over the parietal eminence, the pain does not differ much from that caused by neuralgia of the ophthalmic nerve; and you may remember a case bearing on this point, namely, that of a young woman who lay in bed No. 7, in St. Bernard ward. She complained of an exceedingly acute pulsatile pain, resembling neuralgia in every respect, in the right frontal eminence. On examining her, I found an exostosis there, and swelling of the periosteum of both mastoid processes, which were equally painful.

On close examination, however, there will be found certain characters which enable one to recognise neuralgia properly so called, from intra-osseous pain occurring in syphilis. Thus, ask yourselves how often you have seen true neuralgia of the parietal eminence exactly limited to the point of exit of the small branch which comes off from the outer frontal branch of the ophthalmic. In neuralgia it is easily ascertained that the principal branch is always painful over the supra-orbital notch, whereas it is quite the reverse in osseous pain. Excessive pain is felt on pressing the most prominent part of the bony growth, but by carefully exploring the neighbouring parts, it is found that the pain diminishes in proportion to the distance from the central spot; in a word, the same thing happens as in the case

of a boil, the pain of which diminishes in proportion to the distance from the seat of the boil.

The difference is still more striking if the *tender spinous points* be looked for. You know that, up to this time, there has not been a single case of facial neuralgia under my care, in which the posterior surface of the first two cervical vertebræ was not very tender on pressure; while you saw that this essential sign was absent in the young woman who complained of pain in the frontal bone, simulating neuralgia.

I told you just now that syphilitic bony growths may, by compressing a nerve trunk, give rise to true neuralgia, just as any other tumour. In such cases the pain will not be exclusively seated in the growth, but along the track of the affected nerve. I cannot say whether in such cases there will be tenderness on pressure of the spinous processes, as in other neuralgias, for since my attention has been directed to this point, I have not had the opportunity of seeing a single case of neuralgia due to the presence of a tumour. The absence of tenderness on pressure of the spinous processes is useful again for distinguishing pleuritic from neuralgic pain. You remember a young woman who, in November, 1863, was placed at No. 1, in St. Bernard ward, and was affected with double acute pleurisy. I gave a clinical lecture on her case. On the third day of her getting ill she had, on both sides, amphoric, cavernous respiration, and gurgling, all which disappeared completely at the end of eight days, as she became convalescent. She had pleuritic pain on the right side. I took the opportunity, at the time, of showing you first that the pleuritic stitch generally occupied in the walls of the chest another seat than intercostal neuralgias, for it rarely happens, indeed, that in front it extends beyond a line drawn perpendicularly from the nipple to the abdomen; while intercostal pain, on the contrary, is felt in front of this line, and is diffused over the base of the sternum as well as over the epigastric region. Again, slight pinching and scratching of the skin gave no pain where the pleuritic pain was most sharply felt, whereas the reverse obtains in neuralgia. If, on the other hand, deep pressure was made over the painful intercostal space, it was easily ascertained that the pain was deeply seated, and increased in intensity in proportion to the degree of pressure. Lastly, as in cases of frontal exostosis, the pain corresponded to the inflammation.

All the spinous processes could be squeezed hard without giving the least pain; so that this is an important distinction between intercostal neuralgia and pleurisy; and I cannot therefore admit the views of those pathologists who believe that pleurisy gives rise to a neuralgia, and that this constitutes the stitch in the side which characterises inflammation of the pleura.

Again, in the case of the tenderness on pressure, which is in many cases met with at the outset of tubercular phthisis, when pressure is made on the first intercostal space in the vicinity of the sternum, a pretty sharp pain is always excited on the side where tubercles are deposited; but this pain, which is evidently connected with the chronic pleurisy developed round the upper lobe of the lung, is neither accompanied with tenderness on pressure of the spinous processes, nor of the area of peripheral expansion of the nerves, as you may easily verify in all cases of phthisis. Hence, this differential sign may be of some value in diagnosis.

It is in general pretty easy to recognise neuralgias of syphilitic origin. In most cases they are due to an outward lesion, pretty readily detected, such as an exostosis, periosteal swelling, a node, inflammation of the mucous membrane, ulceration, or necrosis.

§ 4.—*Treatment of neuralgias.—The first indication is, to give relief.—Powerful effects of narcotics.—Later the specific cause is to be combated.—Marvellous effects of revulsion in all its forms.*

It is a well understood thing, gentlemen, that you are not to expect, in our clinical conferences, a complete description of neuralgias, and in some sort a treatise on the subject. As several cases of neuralgia came at the same time under observation, I embraced the opportunity of giving you some general notions on these affections, which are often considered hopeless, both by physician and patient. I have been particularly anxious that these cases should enable you to judge of the effects of the principal modes of treatment which are generally had recourse to in neuralgias.

A general indication is that of relieving the pain, whatever may be the cause under the influence of which the neuralgia has developed itself. There are some cases, undoubtedly, in which the cause may be removed immediately, and the pain disappears with the removal of the cause. Thus, neuralgic pain due to a neuroma ceases at the very moment when the portion of nerve in which the tumour has been developed is cut away; and in the same way, neuralgic pain in the fifth nerve is sometimes instantaneously removed by the extraction of a diseased tooth; and in such cases it is not only the toothache which ceases, but also the reflex neuralgia which had affected the other branches of the trifacial nerve.

But it rarely happens that we can thus get at the cause, and remove it in a moment. When the neuralgia is dependent on a diathesis, a prolonged treatment is needed, and in many cases the cause cannot be removed. In very many instances we fail to cure the rheumatic, the gouty, and the herpetic diathesis,

and we are, *à fortiori*, powerless against tumours of a malignant nature, cancers, and fibrous tumours. In such cases we must, above all, calm the pain, if possible, and either later or at once try to act against the cause which has produced or determined the neuralgia. Even when we can act upon the cachexia to which the neuralgia is due, as in chlorosis, anæmia, and syphilis, the influence of general treatment being essentially slow, our first duty is to moderate the violence of the pain, while trying to modify the general condition of the patient.

It is all the more important to follow this rule, that the neuralgia itself often prevents the general treatment from being successful. For a chlorotic female, for instance, will not bear the steel and the bitters which are so needed in her case, if she be continually a prey to horrible pain, which deeply shakes her nervous system, and disturbs all her functions. Of the means in our power for relieving the pain, drugs which have a *stupefying* influence rank first, such as chloroform, ether, opium, solanaceous preparations. Cyanide of potassium comes next, and only under circumstances which I shall presently indicate.

Next in order, again, come turpentine, irritating applications, acupuncture, electropuncture, faradisation, and warm baths of very prolonged duration. Lastly, specific remedies, when there is a specific cause,—iron, bark, mercury, etc.

This summary indication of remedies which are so numerous and so different, would be of little help to you, if I did not studiously enter into somewhat minute details respecting those various methods of treatment, without which they are apt to lose their power and their opportune application.

When the neuralgia is superficial, and is, for instance, seated in the temples, the forehead, or the scalp, *belladonna* or *atropia*, *applied locally*, is sufficient in a pretty large number of cases. Solutions of atropia have the great advantage of being very powerful, and of not messing the clothes or the skin. I generally use the following formula:

℞. Atropiæ sulphatis . . . gr. v.
Aquæ distillatæ . . . . ʒiij. Solve.

Compresses steeped in this solution are applied over the painful parts, and covered over with a piece of oil-silk so as to prevent evaporation, and the whole is kept in place by a bandage or a handkerchief. The application is renewed several times in the twenty-four hours, and continued for at least an hour each time.

The efficacy of this solution varies in different individuals, so that the dose of atropia should be diminished or increased according to the effect produced. When there is considerable impairment of sight and dryness of the throat, the quantity should either be diminished, or the application be less frequently

renewed. On the other hand, when the absorption of the drug is marked by slight phenomena, while the pain is not relieved, the dose should be increased and the compresses applied almost continuously. When the neuralgia occupies a very limited area, a much more concentrated solution of atropia should be used, namely, three grains of atropia dissolved in about half an ounce of water, with the addition of a small quantity of alcohol; this should be used in drops rubbed on the part with the finger. This is done two or three times in an hour, and a very powerful effect is often obtained in this way. When the neuralgia affects the scalp, and involves the occipital nerve, for instance, the roots of the hair and the scalp should be well wetted, and absorption then takes place with very great facility. A solution of atropia, applied locally, is pretty successful also in neuralgia of the superficial cervical plexus, but is not so useful in intercostal and brachial neuralgia, and loses all efficacy in neuralgia of the lumbar plexus, of the sciatic nerve, and of the abdominal viscera. Yet, you must not think that it is always useless in such cases.

The extract of *Datura stramonium*, and of *Belladonna* may be substituted for the atropia. Formerly ointments made with axunge or cerate were used; but the fatty matters diminished the rate of absorption so much that the treatment was often useless. Glycerine and starch are now used instead, and when the extract is mixed with them, in the proportion of one-third or of one-fourth, a paste of the consistency of an ointment is obtained, which offers the great advantage of spreading easily on the skin and of being soluble in water, so that if a piece of wet lint covered over with oil-silk be laid over it, the skin is placed in the most favourable conditions for absorbing the remedy. As good results are obtained in this way as with atropia, and although the skin of the face and of the throat, and the hair may be a little soiled, the mixture offers immense advantages in regions to which soothing fomentations or poultices may be applied, such as the walls of the chest, the abdomen, and even the limbs. In a word, since this combination has been more frequently used in practice, there has been a greater proportion of cases in which alleviation, if not the cure of pain, has been obtained, whether the pain were merely neuralgia, or were due to an organic lesion, or even a local inflammation.

Opium, in all its forms, is far from being as useful as the preparations of solanaceous plants. The *salts of morphia* offer this immense advantage, however, that they can be applied to the raw surface of the skin, an important method of treatment of which I must speak with some details.

It is not an indifferent matter how the true skin is exposed.

A blistered surface made by means of cantharides is far from giving the same result as one obtained by means of ammonia, and in the latter case the results are not always the same everywhere. When cantharides are used, a morbid process goes on in the skin, which probably lasts a pretty long time after the blister is removed, and impedes absorption to a certain extent. It is not easy for me to tell you the reason why, but it is a clinical fact; and the same dose of sulphate of morphia sprinkled over the surface of the skin made raw by means of cantharides, produces considerably less effect than when ammonia has been used. I entered into very minute details in my Treatise on Therapeutics,[1] on the *mode of using ammonia* for raising a blister, and, on the manner in which the blister should be dressed. The other day, in a young woman at No. 31, who was suffering both from chronic peritonitis and neuralgia of the fifth pair, I raised a blister myself with ammonia, first, in order to show you how it should be done, and secondly, that you might see the rapidity with which salts of morphia are absorbed, and which cannot be credited unless it has been actually seen. I had recourse to the simplest plan. I filled three-fourths of a thimble with very dry cotton-wool well pressed down, and placed in the remaining fourth another piece of cotton-wool steeped in caustic ammonia. I then held the thimble over one temple for about five minutes, and on removing it you could see that the skin which had been in contact with the ammonia was rather paler than the rest, and that all round it the integuments were congested. By passing the finger over the circular mark left by the thimble, the epidermis was seen to move and get wrinkled, thus showing that it was detached. On then rubbing the surface with a piece of linen rather roughly the epidermis was entirely removed, and the cutis vera exposed. I next made a paste of semi-liquid consistency with a drop of water, and one-fifth of a grain of morphia, and laid it over the denuded portion of skin; lastly, I covered the spot with a round piece of oil-silk kept in place by a larger piece of sticking plaster. I shall presently tell you why I had recourse to this kind of dressing.

At the moment when I laid the paste over the raw surface, I asked you to look at your watch, and wait for the first signs of narcotism. I made the patient sit down in order that these signs should be evident. Scarcely had a minute and a half elapsed when she complained of flushings of the head; in another minute, she felt giddy, and lastly, three minutes after the dressing had been applied, she felt such malaise that she

[1] Traité de Thérapeutique (Trousseau et Pidoux), Art. Ammoniaque et Opium.

could not sit any longer: she then laid down, feeling sleepy, and by that time already, as you heard her declare, her pain had diminished markedly. On the following day, as you may remember, the phenomena indicating absorption of the medicine showed themselves with still greater rapidity; but they were long before appearing on the third day, and they could be scarcely detected in the course of it; but when, on the next day, the small sore was dressed, this apparent anomaly was explained, for the sore had almost healed up, and the greater part of the morphia still remained on the surface of the skin.

I called your attention, on the second day, to a rather important point, which would otherwise have been unnoticed. The skin looked raw when I removed the dressing, and yet I told you that there was a thin fibrinous membrane on it, which I then removed by gently rubbing the surface. Bear this simple fact in mind, gentlemen, for if you used the morphia again without removing the false membrane, it would be more slowly and less completely absorbed. You saw at once the reason why I dressed the raw surface as I did on the first day. Had I merely used a strip of sticking plaster or a piece of rag rubbed over with some fatty substance, part of the morphia would have been wasted in the dressing; and the fibrinous secretion of the sore, instead of forming a false membrane, would have soaked the dressing, so that when the morphia came to be used again, the skin would have been seen to be irritated, and much less capable of absorption than when the thin false membrane which has formed underneath the oil-silk is removed.

There is another circumstance which I wish you to remember. When a raw surface made by ammonia is dressed with morphia, the patient feels stupefied two or three minutes afterwards, and the effects which are proper to opium go on increasing for several hours, in a degree which varies considerably according to the age and sex of the patient, and according also to certain circumstances which cannot be made out. Now, if on the first day, the narcotic effect have been moderate, one is tempted to increase the dose on the following day, on the supposition that the system is already used to the opium and will feel its influence much less the second time. But the fact is, and I made you notice it, that the first effect of the remedy is still more rapid than on the previous day, so that it is not uncommon to meet with women who feel perfectly giddy a minute or a minute and a half after the use of the salt; and again, the effect produced is unquestionably greater on the second day, even when the same dose has been used. This is due to a circumstance which may be easily recognised, provided some care be taken. When the ammonia has just been applied,

there is a violent irritation of the true skin, which for nearly an hour is attended with a very abundant secretion of serosity. This trickles from under the dressing, and if you taste it (as I have often done), you will find that it is extremely bitter, from the presence of the morphia, which is dissolved in it. Hence it follows, that a variable quantity of morphia is carried away and is consequently not absorbed; whereas, in the same evening or on the following day, when the false membrane is removed, the true skin no longer secretes any serosity, the whole of the salt remains in contact with the surface of the sore, and the narcotic effect necessarily increases. Hence the rule that, in order to produce the same effects, a smaller dose of morphia should be used the second time. Anyhow, gentlemen, a small quantity of morphia should alone be used in dressing a raw surface made by ammonia, for absorption is nowhere so active as over the skin deprived of its cuticle, and grave accidents may result from the use of high doses, from the first. Never begin, in the case of a woman, with more than one-fifth of a grain; and in that of a man, with more than two-fifths; take care to increase this quantity only when you have ascertained how your patient bears the remedy.

The first effect of the morphia is marvellous: a few minutes are sometimes sufficient to calm an awful pain; and it rarely occurs that great relief is not afforded in a violent neuralgia. But there is a world-wide difference between this and a real cure, and the pain generally returns when the narcotic effect of the drug has passed off. It is necessary then to keep the system under the influence of the remedy for a more or less prolonged period; and a fresh application is to be made in the evening, and twice again on the following day. In that way the pain may, in a great number of cases, be entirely suppressed for some time.

I have already told you that, on the third day, the raw surface made by ammonia no longer absorbs, so that a fresh sore has to be made, in the same manner, in the vicinity of the former, or in another part, according to the intensity of the pain. Care should always be taken that the skin is irritated sufficiently to produce detachment of the cuticle, but never so as to give rise to a very prominent bulla, because otherwise superficial ulceration of the skin would be produced, and absorption rendered more difficult, and on the other hand, a persistent scar would be left behind, a circumstance to be taken into consideration when dealing with the face and other parts exposed to view.

The narcotics should thus be persevered in for eight, ten, or fifteen days, sufficiently long to destroy a vicious habit of the system. When the external application of ointments containing narcotics, of solutions of atropia, or of morphia on blistered

surfaces has failed, still this mode of treatment has not said its last word. There still remains the *hypodermic method*, which, in a great many cases, proves more useful than the others. You know how this is employed: invented by Rynd, it was popularised in England by Wood chiefly, and by M. Béhier in France. With the small syringe which Pravaz invented for the purpose of injecting a coagulating fluid into aneurysmal tumours, a very concentrated solution of basic sulphate of atropine or of sulphate of morphia is injected subcutaneously, as near as possible to the nerve trunk which is the seat of pain. Atropine is generally preferred, and the solution is made with one grain of the sulphate to 100 minims of distilled water: when morphia is used, one grain is dissolved in twenty minims of distilled water. Each drop of the atropine solution is equivalent to $\frac{1}{100}$th of a grain of the salt; and each drop of the morphia solution to $\frac{1}{20}$th of a grain of morphia. Now, as each turn of the handle of the syringe propels one drop, the quantity which is injected may be measured with extreme facility. From four to five, and even ten and fifteen drops of the solution are thus injected: the doses should be small at first, so as to test the susceptibility of the patient, and should be increased by degrees.

This method is chiefly used in deep-seated neuralgias, and although some of my colleagues have praised it almost to exaggeration perhaps, it is of great value, and deserves a place by the one which I have just described in detail to you. The small puncture of the skin frightens pusillanimous persons only, but it offers certain advantages of its own, and I have several times abstained from injecting after having punctured, and this simple acupuncture has sufficed to produce great improvement. I shall presently tell you what we may expect from acupuncture and electropuncture alone, in the treatment of neuralgias.

You are aware, gentlemen, with what difficulty atropine is borne. Some persons cannot take internally a granule containing $\frac{1}{30}$th of a grain of atropine without being poisoned, as it were, or without feeling, at the very least, very uncomfortable sensations in the throat and eyes. I confess that, knowing how much the gastric juice occasionally modifies certain vegetable substances, I expected to obtain, by injecting a narcotic solution into the cellular tissue, much more powerful effects than by having recourse to the ordinary mode of administration. I was, however, mistaken, and I found, to my great surprise, that ten, and sometimes fifteen, twenty drops of the solution of atropine, which were equivalent to $\frac{1}{100}$th and even $\frac{1}{30}$th of a grain of the salt (a dose which would certainly give rise to serious symptoms of poisoning if taken internally), were

borne with the same facility as $\frac{1}{50}$th or $\frac{1}{25}$th of a grain, when taken in the usual way. The same observation applies to morphia, and I must add that the treatment should be persevered in for some time, in order to obtain good results from it.

More than thirty years ago, I thought of a mode of treatment which has always given me more complete results than the endermic and the hypodermic methods, in cases of deep-seated neuralgias, and of sciatica in particular. You probably recollect two men in St. Agnes ward, at No. 8 and No. 14, who were both affected with a very painful and obstinate sciatica. You remember how useless subcutaneous injections of atropine proved to be, although large doses were used and persevered in; a few hours' relief was merely obtained, and at the expense of a feeling of considerable discomfort. Turpentine, also, given in very large doses, produced but trifling improvement; and I then had recourse, with complete success, to a method which I formerly called *hypodermic*, a name which is more appropriate to the subcutaneous method.

In this method, the patient is made to lie on the abdomen, and a portion of the skin of his buttock is pinched up, perpendicularly to the axis of the trunk and over the point of exit of the sciatic nerve. A straight bistouri is then run through the pinched integuments at the base of the fold, and is next made to cut from within outwards. The incision gives scarcely any pain, and, when made in that way, is clean and without tail. The subcutaneous cellular tissue is exposed at the bottom of the wound, and, after having filled this with some dry lint, kept in place by strips of sticking plaster, the patient is left till the next day. Sometimes, however, although rarely, this simple operation suffices not only to give relief, but actually to cure. On the ensuing days small boluses, prepared according to the following formula, are placed in the wound.

℞ Extracti belladonnæ vel stramonii . . . gr. xl.
Extracti opii . . . . . āā
Pulveris guiaci . . . . . gr. lxxx.
Mucilaginis gummi Adragantæ . . . q. s.
Divide into twenty boluses, and dry in an oven.

Each bolus contains two grains each of extract of opium and of extract of stramonium or of belladonna. The guiacum and the gum adraganth only serve to harden the mass, without preventing them from softening and yielding some of their principles.

Two, and even three boluses, should be placed in the wound, and kept there by a piece of sticking plaster, under which, if possible, a small plate of very flexible lead, or several layers of tin, should be laid. A very dry pea should be placed at the same time in the wound, and, on this swelling out considerably during the day, the wound is maintained in a perfectly

open condition. The absorption of the drugs goes on pretty actively, and the narcotic effects may be easily graduated by increasing or diminishing the number of boluses: if necessary, other boluses are prepared, containing a smaller quantity of extract.

One dressing is sufficient in the twenty-four hours, although a more rapid cure is obtained by dressing the wound morning and evening, when less boluses should be used each time. In order to obtain the full benefit of this mode of treatment, the system should be kept constantly under the influence of the drugs. So long as the pain continues, the treatment should be persevered in as above, but when the pain has ceased, a single bolus is laid inside the wound on each occasion with a dry pea; and when all pain has ceased completely for eight or ten days, peas alone are used, as in the case of issues.

I must declare that this mode of treatment has given me better results than any other in sciatic neuralgia, for it combines the action of narcotics and issues. We have seen already the effects of local applications of narcotic substances in the treatment of neuralgias, and I will presently tell you what may be obtained from superficial and deep revulsion. You may understand, therefore, that the combined influence of deep cauterization and of narcotics is followed by better results than the isolated use of either of these measures. This method offers another advantage again, namely, that after all pain has disappeared, there remains a superficial sore, a true issue, which, by being kept open for a few days, or a few weeks, renders the cure certain, and makes it easy, if pain should return in the least, to have again recourse to the boluses, without having to make a fresh incision.

Dr. Lafargue (of Saint Emilion) has recommended a mode of treatment which is of real utility in superficial neuralgias of a slight character, and which consists in introducing under the skin, by a process of vaccination, small quantities of morphia or of atropine, by means of a lancet dipped into a solution of either of those substances. A two-fold action is combined in that process, namely, the effects of irritation of the skin, which is excited by the puncture and the somewhat painful papulæ which are thereby produced (although the benefit derived from this is small, as it is not obtainable by the application of one or more blisters); and secondly, the influence of narcotics, the inoculation probably acting in the same manner as the application of narcotic substances to the exposed cutis.

The application of a solution of *cyanide of potassium* on the skin protected by its epidermis, gives pretty good results when the affected nerves run superficially, as in the face and on the scalp. The strength of the solution should be of one part of

the salt to eighty of water (one drachm to ten ounces). A compress, doubled up several times, is steeped in the solution and applied over the part; a piece of oil-silk is then laid over, and the whole kept in place by a handkerchief. Each application should last from half an hour to two hours, and should be renewed three or four times every twenty-four hours. This method offers great advantages, but it has disadvantages also, of which I must warn you. Pure cyanide of potassium is a caustic, and causes a certain amount of irritation, even when dissolved in eighty parts of water, producing bright redness of the skin, and then a vesicular or papular eruption, which sometimes gives rise to such discomfort that the treatment has to be given up. A greater disadvantage of this solution is that, like strong alkalis, it makes the hair break easily and turns them of a rusty colour, which disappears only when the hair has grown again. I tell you this, gentlemen, in order that you should avoid, if possible, using a solution of cyanide of potassium in parts covered with hair.

Chloroform may be used in nearly the same circumstances as cyanide of potassium, but it should never be applied in a pure state, at least on the face and on parts which are habitually uncovered, because it gives rise to considerable irritation and even vesicates sometimes. It may then act on neuralgias in two ways: by revulsion, in the same manner as flying blisters, sinapisms, and the application of tincture of iodine; and by its sedative power. In order to obtain sedative effects, the liniment should contain from one-third to one-half of chloroform. This simple plan, which may be easily carried out, only succeeds, however, in neuralgias of no great intensity, and especially when they are superficial. I shall presently tell you of what use chloroform inhalations are in the most severe forms of the complaint.

As yet, I have only spoken of the *external application* of narcotics; but their *internal administration* is also of unquestionable utility. Solanaceous preparations, and opium, in its various forms, either separately or in combination, have always been, and will always be, powerful remedies against neuralgias. By a patient and intelligent administration of these drugs, results may be obtained which can scarcely be expected beforehand. The dose is of cardinal importance, and it is impossible to lay down precise rules what it should be, for it must vary, according to the duration and intensity of the disease, and according to the manner in which the remedy is borne by the individual. In general, however, no fear need be entertained as to keeping the patient fully under the influence of the drug, as shown by the manifestation of the physiological effects proper to each of them. Chloroform and ether, used as anæsthetics,

render marked services also in the treatment of neuralgias. You know the good effects of *chloroform* inhalations in hepatic and nephritic colic, even when a calculus has got inside the excretory duct. You remember the case of that woman who suffered from biliary calculi, and whose pain was immediately relieved, even when the paroxysm was at its height: she suffered also from a very acute intercostal neuralgia, which was immediately relieved as soon as she got under the influence of chloroform. A youth, from Poissy, affected with gravel, was often seized with awful nephritic colic, whenever a large pyriform calculus tried to pass through the ureter. The pain, however acute it might be, was instantly relieved by chloroform inhalations, and the patient could be then placed with his head downwards and squeezed over the region of the kidney, so as to push the calculus back into the pelvis, when the attack ceased. In this case, the inhalation had to be pushed on to the stage of unconsciousness; but in order merely to relieve nephritic or hepatic colic, it is sufficient to induce a state of semi-unconsciousness, as when chloroform is used during labour.

Although the unconsciousness produced by chloroform or ether be transitory only, the narcotic effect induced continues for a pretty long period, and it frequently occurs that neuralgic pain yields or becomes very bearable at least, during half an hour or an hour even. It happens pretty frequently also that the anæsthetic stops the paroxysm completely, especially when the neuralgia assumes a paroxysmal character.

When chloroform or ether inhalations are used against neuralgic pain, no apparatus is needed, nor even a handkerchief. The patient makes a sort of cone with his hand, by bending his little finger completely into his palm, and his other fingers less completely: the cone is open between the thumb and index finger, and as the fingers are slightly separated from one another, air passes through easily. From ten to twenty minims of chloroform are dropped into the palm of the hand, which is then held before the nostrils, while the patient draws a deep breath. A single inspiration, when well made, is sufficient to cause giddiness, which is in some cases violent enough to make the patient fall if he happens to be standing at the time. Many among you who have tried this simple plan have been often compelled to sit down, in consequence of the deep impression produced by the chloroform inhaled in this way. The inhalation may be repeated once or twice, successively, and as frequently afterwards as the return of the pain requires it. I need scarcely add, that the quantity of chloroform or of ether thus inhaled is too small ever to give rise to any accidents.

The internal administration of quinine is often of very great service in the treatment of neuralgias. When the affection is

due to the influence of marsh miasmata, bark cures it, as it does all the other manifestations of the same specific cause; but, even when the neuralgia is not due to this cause, quinine exerts a powerful influence on the disease. It acts perhaps in the same manner as in rheumatism and in gout, and this is all the more probable because the neuralgia is very often the expression of a rheumatic or gouty diathesis. In such cases, quinine should be given in larger doses than in intermittent fever, and these larger doses should be continued for a longer period.

Iodide of potassium also cures certain neuralgias which have evidently nothing to do with syphilis.

Spirits of turpentine have been long vaunted in the treatment of neuralgias, and Récamier and Martinet have dwelt on the advantages which may be obtained from its internal administration. But the disagreeable taste of turpentine prevented its becoming a popular remedy. The patient had an unsurmountable aversion to it after a few days, and gave it up. It had another and a somewhat serious disadvantage, namely, that it irritated the mucous membrane of the fauces and œsophagus on its passage, and to such a degree as occasionally to excite acute pain and vomiting. The irritating action of the drug was also felt in the stomach, and the manner of administering it added to the drawbacks. Some of these were avoided by administering it per rectum, and this method was particularly advised in sciatica; but the extent of absorbing surface was insufficient on the one hand, and on the other the rectum soon became intolerant of the drug, so that this useful plan had to be given up.

All these disadvantages are now removed, by the administration of turpentine in capsules, which thus reaches the stomach without being tasted, and without irritating the pharynx and œsophagus. On the other hand, the medicine should be taken at meal time, a precaution to which I attach great importance, and by the aid of which turpentine and many other drugs are easily borne. It is of the highest practical importance that irritant remedies which are administered by the mouth do not get in contact with the unprotected membrane of the stomach. Preparations of iron, quinine, turpentine, iodine, and mercury, and many other therapeutic agents possessed of irritant properties, cannot, in most cases, be taken without inconvenience, for the sole reason that they are administered between meals; when taken with the food, they do not irritate the mucous membrane, while their specific power is not modified in the slightest degree.

You see me, therefore, gentlemen, prescribe turpentine in all my cases invariably, and you hear me give strict injunctions to the sister that the patient should take the capsules at his meals,

and you witness yourselves the facility with which large doses of the drug are borne, while it very rarely happens that unpleasant symptoms compel me to suspend its administration.

Lehuby's capsules, which contain from eight to ten drops of turpentine, are those generally used in the Paris hospitals. I give from four to five or six of these capsules, twice a day (that is, from 100 to 120 drops a day), and this is generally enough, although this dose may be doubled, or trebled, without inconvenience. The medicine is continued for six or eight days in succession; I then intermit it for four or five days, after which I resume it, and so on for several weeks.

I must, in justice, however, declare that turpentine fails in half the cases of neuralgia, although it is a good remedy, of which practitioners should avail themselves. Quite recently you saw me on several occasions prescribe it in the case of a woman, lying in bed No. 29A, in St. Bernard ward, who was affected with violent neuralgia of the trifacial and of the nerves of the stomach. The facial neuralgia disappeared first, and the gastric pain a few days later. Some of you must surely have thought it strange that I gave turpentine in large doses when the stomach seemed to be so affected. But a moment's reflection enables one to understand easily that neuralgia of the plexus which supplies the stomach, does not indicate inflammation of the mucous membrane, and that there is no reason why turpentine should not be given in such a case, as in hepatic or intercostal neuralgia. It is a fact, however (and I shall not attempt to explain it), that gastric neuralgia is in general more easily and more surely cured by turpentine than other neuralgias.

Irritant applications are of great service in the treatment of neuralgias, although much less so than is believed by most medical men. From Cotugno, who recommended, with such confidence, the application of *blisters* in sciatica, over three selected spots, the buttock, the head of the fibula, and the malleolus externus, down to Valleix, who regarded blisters almost like a panacea, practitioners have used them constantly in the treatment of neuralgias, although I think that they have owed their favour to the extreme facility with which they can be used, for I cannot believe in the exaggerated praises which have been accorded to them for a century.

When the neuralgia is recent, and is apparently connected with rheumatism, it is often removed by irritating applications to the skin, such as painting it with *tincture of iodine*, or covering it with a *mustard poultice*, or rubbing it with *croton oil*, or with an *ammoniacal ointment*. But when the disease is chronic, and may be reasonably ascribed to a diathesis, as the gouty, the herpetic, the chlorotic, the aguish, or the syphilitic, the relief procured by the irritant applications is only temporary, and

lasts for a few days, or even a few hours only. Yet, by having recourse to powerful revulsion, proportionately to the old date and the violence of the neuralgia, results are sometimes obtained which less energetic treatment does not procure; and it is thus that *moxas* and *flying cauteries* are of unquestionable utility.

I often mentioned in my lectures, when I was professor of therapeutics in the Faculty of Medicine of Paris, that the public executioner at Lyons formerly enjoyed the reputation of curing sciatica. He wrapped the whole lower extremity in a large pitch plaster, and the eczematous eruption, which soon showed itself after this from the hip to the toes, acted with a power which a less heroic treatment could not exert.

You have seen me, within the last few years, try in the hospital an instrument invented by a man who does not belong to our profession, and which he has termed the *awakener* (*le réveilleur*). It consists of a stem, bearing at its extremity numerous small steel points which cannot penetrate deeper than a millimetre (about half a line). The stem has a bell spring attached to it, which, on being touched, drives the points into the skin with extreme violence and vivacity, thus producing multiple and very superficial acupuncture. The skin is next anointed with essential oil of mustard, dissolved in some olive oil, which soon gives rise to great local irritation, more painful than is generally caused by mustard alone.

This method has been chiefly used in the treatment of rheumatism, but it is really of some service in cases of neuralgia. It is analogous to *acupuncture*, which you have seen me use successfully in the wards on several occasions. For that purpose I use steel needles, which I soften, by heating them to redness in the flame of a candle. The head of each needle is protected by a small piece of sealing wax, and one or more of them are pushed into the painful regions, without taking care to avoid the nerve trunks. They should be left in for ten minutes, and even for an hour, and the operation should be repeated two or three times a day, for several days, and for a few days after all pain has ceased.

Electro-puncture is a more painful process, but it is of still greater utility. In 1863, I saw, in consultation with Dr. Demarquay, an old gentleman of 65 years old, who, after an attack of zona on one side of the forehead, had for more than a year been tormented by pain which almost drove him mad. Quinine, in large doses, local irritant applications, and narcotics, given internally and applied externally, had failed. The patient obtained admission into the *Maison municipale de Santé*, and came under Dr. Demarquay's care. This skilful surgeon had recourse to electro-puncture, and in a few sittings the pain disappeared, but only for a time, however, for it showed

itself again with its former intensity at the end of a month. Dr. Duchenne (de Boulogne) conceived the happy idea of trying very powerful *faradization of the skin* in obstinate neuralgia; and this plan, which is extremely painful, sometimes produces marvellous effects. It is not uncommon to see the horrible neuralgic pain of *angina pectoris* yield to its influence. Epileptiform neuralgia, which is such an exquisitely painful and a cruelly incurable affection, is sometimes quickly modified, but not *cured*, by this method. But, although the violence and persistence of neuralgic pains induced patients to submit to cutaneous faradization, few of those who have been but recently affected can be persuaded to adopt a method of treatment which causes such insufferable pain.

I have often shown you the extraordinary effects following the *application of heat* to joints that are the seat of chronic painful engorgement, by means of sand bags heated to as high a temperature as the patient can bear without being burnt. This plan succeeds very well also in superficial neuralgias, as those of the scalp, the face, and the neck; and even when the pain affects limbs in their continuity. You have seen me, on several occasions, wrap in these sand bags the heads of patients, for twenty minutes at a time, and repeating the process twice a day.

This plan is certainly not so efficacious as those which I have already described to you, although it proved very useful in some cases in which every other treatment had failed completely.

There is another method, to which I have had recourse several times in my life, but in which I have found few imitators, however much I have said on the subject. I allude to *division of the temporal and of the occipital arteries*, with the view of curing obstinate neuralgias of the head. Division of the painful nerve had already been recommended in epileptiform neuralgia, and, in the majority of instances, it was impossible, except by actual dissection, to divide the nerve without also cutting the artery which usually accompanies it. I have given you already, in my lecture on epileptiform neuralgia, my opinion of this operation in such cases; but after having on several occasions divided the artery, in obstinate cases of the ordinary form of neuralgia, I obtained such immediate results that I in vain ask myself to this day how the method acted. I tried it for the first time in 1833, in the case of a lady, about 30 years old, who had been for more than 10 years afflicted with temporo-facial and cranial neuralgia, of excessive violence. I had tried no end of remedies; and, as a last resource, I determined on dividing the temporal artery above the zygomatic arch. I wrapped the blade of a bistouri in a piece of sticking plaster, leaving only a third of an inch of the point uncovered, and, holding the instrument like a

pen, I pushed it through the tissues, quite close to the ear, perpendicularly, and on reaching the bone, I went on cutting until there was a gush of arterial blood, always keeping the knife parallel to the upper edge of the zygomatic arch. The neuralgia ceased as soon as the section was made. As my object was not to draw blood, I immediately applied a compressing bandage, which was removed twenty-four hours afterwards. The neuralgia was cured for a rather prolonged period, and, although it returned subsequently, I still regard the case as a very successful one. Division of the occipital artery is not less efficacious in the treatment of neuralgia seated in the posterior region of the head; and it is often found necessary to divide both arteries, an operation as easily performed as it is free from inconvenience. I said just now that I could not account for the suddenness of the improvement following division of the arteries. I am well aware that these vessels, especially the occipital, are accompanied by nervous twigs of some importance; but although I understand how the pain ceases in parts supplied by nerves included in the section, I cannot understand the complete cessation of pain in the greater number of nerve twigs, which a moment before caused such acute pain, and which have no apparent connection with the cut branches.

The following case is a most curious instance in point:—

I was asked by Dr. Mathieu, to see a man about 30 years old, in the *rue Neuve Saint-Méry*, who was suffering from acute encephalitis. The poor man complained of excessive pain in the head, and uttered fearful cries; narcotics, employed internally and externally, had failed to relieve, and bleeding had proved as ineffectual. I advised division of the temporal artery, and operated at once; the relief was instantaneous, although scarcely a spoonful of blood had been lost. The patient was admitted into my wards in the hospital, and after his death, an abscess was found in his brain. I have related this case, gentlemen, merely to show you the utility of dividing the artery, even in cranial neuralgias that are symptomatic of the gravest lesions.

It may be asked whether the rapid improvement which follows division of the artery and of the nervous twigs which accompany it is the result of a mental impression, in some respects analogous to the impression produced in toothache by the sight of a dentist's instrument; but I shall not attempt to answer the question. Yet, when we see facial neuralgia and sciatic neuralgia itself cured by division or cauterization of the lobule of the ear (and there are now pretty numerous cases of the kind on record), how can we account for the beneficial influence due to division of the temporal and occipital arteries in neuralgias of the head?

As yet, gentlemen, in the long list of remedies which I have enumerated to you, I have only had the element *pain* in view, and I have not taken into account the cause to which the neuralgia was due, except when this cause was immediately cognisable, as in toothache dependent on a carious tooth, in neuroma, in wounds or in injury to a nerve-trunk, &c. But when the cause acts on the whole system, as syphilis and chlorosis for example, we can and we doubtless should calm the pain as quickly as possible, but the chief aim of treatment should be to remove the cause. Otherwise the neuralgia is only relieved for a time, and returns after a brief interval; for when it disappears easily under the influence of the various measures which I have described to you, it is brought on by causes which manifest themselves by transient phenomena only, as rheumatism, for example.

Neuralgia, of syphilitic origin, is of rare occurrence, except when there is a local lesion, such as exostosis, periostitis, nodes, &c., and the general rheumatoid pain, which is sometimes present in cases of constitutional syphilis, is probably due to irritation of the spinal cord. It is often brought on again by an acute inflammation, and by ulceration of mucous membranes, as in syphilitic coryza and otitis. All these neuralgic phenomena are rapidly removed by specific treatment, but if necrosis be present, mercury and iodine will, of course, fail, and will only influence the disease which brought on the necrosis. I have told you the signs by which you could recognise neuralgias of syphilitic origin, and you have seen, in the wards, how quickly a specific treatment did away with the pain. You remember the case of a woman, who, in June, 1863, lay at No. 7, in St. Bernard ward. The neuralgia was worse rather late in the evening, and was better in the morning. I gave her two grains of calomel a day, to be taken in doses of the tenth of a grain in the course of the day, and on the third day of this treatment, as soon as the gums began to swell slightly, the pain ceased almost entirely. The solution of Van-Swieten (*Liquor corrosivi sublimati*) was then substituted for the calomel, and subsequently, iodide of potassium was administered.

Calomel, in divided doses (*fractâ dosi*) is the preparation which I always use when I want to act quickly on the system. I have powders made up with $\frac{1}{10}$th of a grain of calomel and from 2 to 4 grains of sugar; and the patient takes ten such powders every day at nearly equal intervals, for three, four, five, or six days. It rarely happens that the gums do not begin to swell by the third day; when they do, five powders only are given, instead of ten. When the pain is relieved, I prescribe chlorate of potash to cure the mercurial stomatitis, and I next give the solution of Van-Swieten for a month or

two, finishing afterwards with iodide of potassium. The effects of the treatment manifest themselves immediately; from the first night, the pain is often diminished, and it is of rare occurrence that it is not perfectly bearable after three days of this treatment. Any exostosis which may be present does not, of course, disappear in that time, but it immediately becomes less tender on pressure, and afterwards disappears slowly.

I am aware, gentlemen, that iodide of potassium does real service in such cases, and you have seen me administer it with great success on several occasions. But I must state, that although it is superior to mercury when administered in the ordinary way, it is infinitely less powerful than calomel given according to the above method. As to intermittent neuralgias, which have been specially termed larvated fevers when apparently due to paludal influences, they are cured by preparations of bark in pretty large doses, larger in general than in ordinary intermittent fevers. But you must not think that intermittence and perfect periodicity in cases of neuralgia are proof positive that the disease is of paludal origin, for I have told you of cases in which a grave organic lesion gave rise to perfectly periodic neuralgic pain, and in which bark failed.

When the neuralgia returns in multiple paroxysms every day, even when these paroxysms are periodic, quinine exerts very little influence. It possesses more power if there be but one paroxysm, while it is a sovereign remedy, as it were, when the paroxysms are tertian or quartan, for these forms, when periodic, are more certain proofs of the existence of paludal influence in the case. Yet, gentlemen (and I cannot account for it), even when there is not the least suspicion that the same causes which bring on intermittent fever have been at work, quinine, in large doses, exerts a powerful influence on neuralgia, even when the attacks are not intermittent, and, *à fortiori*, when they are intermittent and periodic. Hence you see me, in most cases, have recourse to quinine first, and try other remedies only when it has failed.

LECTURE XVIII.

CEREBRAL RHEUMATISM.

Cases of Cerebral Rheumatism occurring in a drunkard and in a woman who had been insane.—The Cerebral Symptoms are generally due to individual predisposition.—Of Delirium in diseases in general.—Six forms of Cerebral Rheumatism: the apoplectic, the delirious, the meningitic, the hydrocephalic, the convulsive, and the choreic.—These divisions are somewhat artificial.—Description of these forms.—Nature of Rheumatism.—Meningitis rare; symptoms and lesions of this affection generally absent.—The Cerebral Phenomena are not the consequence of metastasis, but are generally owing to some morbid cerebral predisposition, such as previous habits of drunkenness, or some former neurosis.—They are not brought on by the administration of sulphate of quinine.—Treatment.

Gentlemen,—You could see a few days ago at No. 16, in St. Agnes ward, a remarkably robust man who was suffering from acute articular rheumatism.

When he was twelve years old, he for the first time fell ill of that complaint, which then affected his lower limbs chiefly, and lasted three months. Six years later, he had a second attack, when all his joints were involved, and for about three months again. At the age of twenty-one, he had a third attack, during which all his joints were successively affected, and which lasted four months. It would be difficult to meet with another case in which the rheumatic diathesis was more marked; and yet the patient declares that he has never had anything the matter with his heart, has never had palpitation, shortness of breath, or œdema. But you shall hear by and by that his heart was seriously affected, and that Dr. Bouillaud's law again proved true in this case.

Twelve days before admission, this patient felt vague pain in the small finger-joints, without any marked fever or malaise. Fever next set in, the left wrist swelled, and grew very painful, and the malaise became general. On the day of his admission, February 19, his fever was pretty sharp; his pulse was 118; his skin perspiring, the left wrist very much swollen, together with the synovial sheaths of the extensors, and of the long abductor of the thumb. The small joints of the carpus were painful. Both knee-joints, especially the left, seized since the previous day only, were painful also. The right knee, which

had been affected for two days, contained a small amount of serous fluid.

I mention all these details in order that no doubt should exist in your mind as to the case being one of acute articular rheumatism, and that you may follow the migration of the symptoms.

A very rough systolic bellows-murmur and a soft diastolic one were also heard over the base of the heart, and could be traced into the large arteries.

From the frequency of the pulse, the heat of skin, the intense thirst, and the general aspect of the patient, I prognosticated a severe attack of rheumatism, of prolonged duration. I prescribed a scruple of sulphate of quinine, and in the evening my clinical assistant cupped the præcordial region in six different places.

On the following days the pulse remained as frequent, but the state of the joints changed; on the 21st, all swelling of the left wrist had disappeared, but there was still redness of the synovial sheaths of the wrist and the hand; on the 22nd, the tibio-tarsal articulation and the right foot were painful, the left hand and wrist were free, but the right hand was affected; on the 24th, the lower limbs were free, but the right hand was still red and swollen; the elbows were free, while the shoulders were painful.

The patient felt much better, and hoped to be soon able to eat. For the previous two days, the dose of quinine had been increased to two scruples; on the 22nd the patient took thirty grains of quinine.

On going round on the evening of the 24th, my clinical assistant observed no unusual symptom; the pain in the joints had diminished, and the patient was very much pleased with his condition. An hour later, however, he complained of not being able to see, and shortly afterwards he began to vociferate, called out "Thief!" rushed out of bed, and fell down. On being put back to bed by two attendants, he struggled with them, exhibiting considerable strength, and then, dropping back, died. All this took place in less than a quarter of an hour.

When the body was examined, there was found pretty marked injection of the whole of the pia-mater covering the brain, but the meninges were nowhere thickened, and nowhere adherent to the grey matter of the brain. There was not a trace of effusion into the sub-arachnoid space. The choroid plexuses were not appreciably redder than they normally are. There was no intra-ventricular effusion. The brain was remarkably healthy, and thin sections of it showed that it was not more vascular in one point than in another; there were not even the

interstitial puncta which are sometimes observed when there is meningeal injection. The corpus callosum, thalami optici, corpora striata, cerebellum, and medulla oblongata were of firm consistency, and exhibited no alteration whatever. In short, it would have been difficult to see a more normal brain in aspect and texture.

The basilar and cerebral arteries were perfectly healthy, with unaltered walls; no coagulum could be seen which could be referred to thrombosis or embolism.

As to the cardiac lesions, I need not dwell on them, but will merely state that the bruits heard during life depended, as had been diagnosed, on a double lesion of the ventriculo-aortic orifice, constriction of the aperture, and incompetency of its valves. Contrary to the patient's assertion, however, these lesions were of very old date, although they had not given rise to functional disturbances. There was intimate adherence of the whole of the pericardium to the heart. The heart was very large, especially on the left; its cavities were empty. There was congestion of the lungs, almost amounting to that found in asphyxia. The kidneys were large, and of a violet hue. The liver was red, and of a very large size. There was nothing worth noticing in other organs.

There was no trace of effusion in either knee; the synovial membrane was not in the least injected, except to a very slight degree in the outer cul-de-sac of the left knee. There was no redness of the synovial membrane, nor any effusion into the wrist-joints, or the other articulations which were affected on the preceding day.

I do not think that any one of these details is superfluous. The present subject is a controverted one, and raises many questions of doctrine; for it may be asked whether, in cerebral rheumatism, there be metastasis from the joints to the brain, mere functional disturbance of the brain without any lesion, or rheumatic meningitis? You may now imagine what interest attaches to the pathological appearances found in such instances.

The subject of this case, then, had previously suffered from three attacks of acute articular rheumatism, which had each lasted from three to four months, and had left persistent marks on the pericardium and the heart. The fourth and last attack was only eight days old, when the state of the articulations and the general condition suddenly improved, while soon afterwards cerebral symptoms developed themselves, beginning with slight disorder of vision, and ending in delirium of only a quarter of an hour's duration, followed by sudden death.

On examination of the body, slight injection of the meninges is alone found to account for the brain symptoms. The case

was certainly one of acute articular rheumatism, and the symptoms which preceded death were unquestionably those of cerebral rheumatism. But they had made their appearance so suddenly, and so taken us by surprise, that, after my attention had been thus aroused, I determined on investigating the case further. I then got the information that the patient had been a hard drinker, or, in other words, a drunkard. He could drink largely without getting drunk, but his companions said that he was *stupefied* by drink. For three months previously he often had at night attacks of dyspnœa and nightmares. His previous history, therefore, told a sad tale of drunken habits, and his brain was in an unfavourable condition when rheumatism attacked him for the fourth time. We shall see, presently, what conclusions can be drawn from this respecting the etiology of cerebral rheumatism. In the meantime allow me to call your attention most particularly to the fact that this patient was a hard drinker, spending his days in a chronic state of inebriation, and that for some time past he was subject to nightmares, and lastly, that he complained of mistiness of vision a few minutes before his fatal seizure.

I pass on to another case, that of a woman aged sixty-three, who is lying in bed No. 2. She is a charwoman and porter, so that she may be a little addicted to spirit drinking. She relates her story as follows :—On the Sunday previous to her admission into hospital, she had, after doing her work, gone to Notre-Dame to mass, but she could not follow the service as usual, and did not understand it, and at the same time she felt an acute pain in her right shoulder. After church she went to a house to do some work as charwoman, although she felt queer. She did part of her work in a mechanical manner, and then sat down in a stupid state in a dark corner of the kitchen, where she remained silent and motionless. Her employer kindly sent her home in a cab. On her way there, she complained of acute pain in her right shoulder, and was conscious of a singular undefined change which had come over her mind. She got into bed, slept well, and woke the next morning speechless. She wanted to drink, but could not make her husband understand her wish; she pointed to the water-bottle with her left hand, but she could not even make those elementary gestures which we would use if we wanted to ask for drink from a person who did not understand our language. She evinced impatience, as aphasic patients generally do, at her inability to speak and gesticulate, and at others being unable to understand her meaning. She had then in her mind a distinct wish to show her anger by calling her husband a bad name, but although she had a definite idea of this in her own mind, she could not speak out. During the whole of Monday and part of Tuesday,

she had not the least power to articulate a single word or to make an intelligent gesture. A medical man was called in, who ordered a few leeches and a purgative. In the course of Tuesday evening she began to speak, but in a sputtering manner, and when she was admitted into the hospital on Wednesday, she spoke in the same sputtering way, although she could pretty distinctly relate how her complaint began. She complained on that day of violent pain in the right shoulder and the left knee.

As her mental condition interested me very much, I pressed her with questions, whether she had suffered from any nervous complaint at some previous time, and she at last told me that she had been rather *nervous* lately, especially since the revolution of 1848. Rents were with great difficulty collected from small lodgers, and one of these even threatened to shoot her husband, on the latter claiming the rent. She was so frightened that she became quite mad with fear, and that she had to be taken to the Salpêtrière three days afterwards, and remained in that hospital for thirteen months, in a state of fierce mania. Now, I maintain that the brain of this woman must have been in an abnormal condition to account for her becoming insane after a violent altercation, and especially for her remaining so long in a state of furious mania.

At this present moment the patient is feverish, her tongue is white, and she feels sick whenever she tries to sit up in bed. Her right shoulder and elbow and her left knee are painful, but they are neither red nor swollen. There is nothing wrong about the heart or lungs. The most remarkable circumstance about her cerebral condition is an irresistible tendency to doze, exactly as if she had been struck with apoplexy. She begins a sentence well, but by degrees speaks less and less intelligibly and rapidly, then stops and drops off to sleep. When she is shaken sharply, she wakes up, looks around with wondering eyes, answers questions clearly, but soon dozes off again.

Thus, gentlemen, the subject of my first case was a hard drinker, who suffered habitually from nervous symptoms traceable to a morbid cerebral condition, while the patient in my second case is an extremely nervous and somewhat insane woman. There was, therefore, in both these cases, a predisposition to the manifestation of cerebral symptoms in the course of rheumatic fever. But before I proceed any further, I wish to make a digression which will enable me to bring before you my views on the various modes of manifestation of delirium, and on the variable significance of this psychical disorder.

You know already from experience that some individuals rave for the least thing, and in order to illustrate how far this predisposition to delirium may be pushed, I will relate to you the

following case. A few years ago, while making a post-mortem examination at the Hôtel-Dieu, both my clinical assistant and I pricked ourselves. My assistant, whose mother was of a nervous temperament, and who had himself been a sleep-walker as a boy, became affected with boils and with very serious nervous symptoms, occasionally even with fearful delirium. Five or six days after I had wounded myself, I had at the site of the wound a carbuncle which gave me great pain, but brought on no fever, and I then had a succession of boils, unattended with fever or delirium. Thus, one and the same cause, namely, a prick inflicted under the same conditions, gave rise to exactly similar anatomical lesions, but to general reaction, which was perfectly different in the two cases, and very manifestly because of the difference in the amount of vital resistance in the two subjects. This is the condition to which the ancients have given the oft-derided name of idiosyncrasy. Thus again, how many individuals do we meet with who are delirious when they have the least fever, and how many children are seized with convulsions when they get at all feverish.

Febrile delirium generally comes on at the *onset* of diseases. Both delirium and convulsions are then correlative phenomena of the rigor; but it must be added that there are certain acquired predispositions. Thus, Dupuytren called attention to the nervous delirium following upon injuries, and justly compared it to *delirium tremens*. Now, this delirium occurs in individuals addicted to spirituous liquors, without there being any relation between the lesion and the intellectual disturbance, for it comes on after any kind of injury as well as after the most skilfully performed surgical operation. You are aware, also, how frequently delirium complicates pneumonia when occurring in drunkards. In the former case the injury, in the latter the inflammation are the determining cause which brings on, with the aid of the existing predisposition, the mental disturbance which is the dreaded prelude of a fatal termination.

Hereditary influence plays also a most important part in the production of nervous disturbances; and hence it is we frequently find that women whose mothers were insane or of a nervous temperament are seized with eclampsia during labour.

As to the prognostic value of delirium, it chiefly depends on the nature and intensity of the disease in the course of which it shows itself. Take, for instance, typhoid fever and cholera, on the one hand, and scarlatina and measles on the other.

Stupor and delirium are, as it were, normal symptoms of typhoid fever, which always presents nervous phenomena, such as sleeplessness, stupor, vertigo, weakness, and delirium. These

symptoms appear very simple to you then, because they form part and parcel of the complaint; and you take them into account merely in order to confirm your diagnosis. But if they show themselves in the course of a disease of which they are not usual accompaniments—in articular rheumatism or pneumonia, for instance—they at once excite anxiety and alarm.

See what happens in cholera, in the most virulent form of the complaint even. The intellect is unimpaired; and although the patient may shriek from the pain of the cramps, he gives rational answers to questions that are put to him. The brain, therefore, is not involved until the so-called typhoid stage of the disease sets in. Take, again, a case of acute peritonitis, when the whole of the abdominal cavity is involved, and the inflammation spreads by contiguity to the intestinal walls themselves; no delirium, no nervous symptoms will show themselves. Such phenomena, therefore, do not depend on the seat of the disease, but on its nature. I must add that, while speaking of cholera and typhoid fever together, as abdominal diseases, for the sake of illustration, I followed the usual custom, although it is a bad one.

Let us now examine the relation of delirium to diseases of the skin, and to eruptive fevers, which are no more cutaneous affections than cholera and typhoid fever are abdominal diseases.

In scarlatina, delirium is, as it were, the rule, as it is in typhoid fever; but not so with measles. If, therefore, delirium should come on about the fifth or sixth day of an attack of measles, it should excite alarm, because this nervous symptom does not form part of the natural evolution of the disease. In some cases the delirium is proportionate to the intensity of the disease, although nervous symptoms do not constitute part of the complaint. Thus, discrete variola is not attended with delirium, while confluent variola is nearly always so. Delirium also occurs in erysipelas of the face, when the face and scalp are simultaneously involved.

The usual course of a complaint should therefore be carefully considered before a prognosis is established. If nervous symptoms be proper to the disease, they should give rise to no great anxiety; but if they be of unusual occurrence, they should be taken into serious account. Thus, delirium occurring in a case of lead poisoning shows that the brain is affected, and compels a modified prognosis; while eclampsia supervening on albuminuria points to urœmic poisoning, and, therefore, to a grave affection.

Let us now examine the relation which exists between nervous symptoms and rheumatism.

Articular rheumatism has no great tendency to develop cerebral manifestations: bear this well in mind. However intense the fever

and the pain may be, this complaint does not usually give rise to ataxic phenomena, to delirium, or to somnolence; the intellect is unimpaired. And yet some cases do occur in which the rheumatism is complicated with brain symptoms, occurring independently of the intensity of the disease, of its gravity, as well as of its extent.

Recall to mind the two cases which you saw in my wards, and which suggested this lecture. The woman lying at No. 2 was suffering from an extremely mild attack of rheumatism, which had scarcely given rise to any marked degree of fever. The intensity of the disease had, therefore, nothing to do with the development of brain-symptoms; and yet, from the second day of the attack, symptoms somewhat apoplectiform in character began to show themselves, giving rise at first to aphasia of forty-eight hours' duration. The man at No. 16 was suffering from articular rheumatism, which was well marked, but not excessively so; the joints that were simultaneously affected were not many, the fever was moderate, and yet formidable brain-symptoms supervened, which carried him off in less than an hour. But this patient was a hard drinker, habitually stupefied by excesses in drink; while the woman I spoke of just now was of an exceedingly nervous temperament, and had been insane. Bear these facts well in mind, because they throw, in my opinion, the greatest light on the etiology of cerebral rheumatism.

Be careful also not to confound such cases with others in which brain symptoms occur constantly, as the consequence of a true typhoid state, such as the purulent arthritis of pyæmia, or of puerperal fever. You may recollect a man who was lately at No. 2, in St. Agnes ward, and on the front of whose chest a heavy bag had fallen, while he was engaged in unloading a cart. He complained of an acute pain in the right flank on the day of his admission, where deep fluctuation could be felt. Two days afterwards his knees swelled, and next his wrists and shoulders. Tremor of the lips, carphology, and delirium then came on, so that I diagnosed pyæmia and secondary arthritis. On examining the body after death, I found an enormous collection of pus inside the chest, and inflammation of the joints which had been attacked. This latter condition had given to the case a semblance of rheumatism, but it was not an example of ordinary acute articular rheumatism. I could not repeat it too often that in this latter complaint brain-symptoms are the exception, while they are the rule in infectious or purulent diseases.

Before I describe to you the symptoms and the forms of cerebral rheumatism, I will first tell you of the premonitory symptoms which may precede it. In some instances there are

none, and they were certainly absent in the case of the woman at No. 2; while in that of the man at No. 16, they consisted in a slight disturbance of vision, which lasted a few minutes. This was a premonitory symptom, however short its duration may have been; in some instances, it has preceded by a day or two the occurrence of brain-symptoms.

It has been also said that an exaggerated anxiety shown by the patient points to a mental condition which should put us on our guard, because acute articular rheumatism does not, in general, alarm the patient. Hallucinations and stupor are symptoms, therefore, which indicate the possible supervention of some cerebral complication.

Dr. Vigla and other observers have also mentioned excessively copious perspiration, and the presence of a miliary eruption as premonitory symptoms. But profuse perspiration is of usual occurrence in rheumatic fever, and this does not indicate that brain-symptoms are imminent. And as the miliary eruption is only the result of the perspiration, it cannot be regarded as a premonitory symptom.

Now, what are the forms of cerebral rheumatism? Six forms have been admitted: 1st, the *apoplectic*; 2nd, the *delirious*; 3rd, the *meningitic*; 4th, the *hydrocephalic*, described by Dr. Marrotte; 5th, the *convulsive*; and 6th and lastly, the *choreic*, of which I have related instances to you. All these forms, in my opinion, are mere modifications of the cerebral condition, and are only required for the sake of description. They are in reality an expression of the same cause, and of the same anatomical lesion, if there be one, and they no more deserve to be regarded as distinct species than the delirious or convulsive form of typhoid fever or of scarlatina.

I will now review successively these various forms, and I will begin with the *apoplectic*. Older authors have admitted it already, and it is mentioned by Storck, Musgrave, and Sauvages. But to what confusion did not the term *apoplexy* give rise in those days? All cases of sudden death were ascribed to it. So that we are justified in believing that the term apoplectic cerebral rheumatism was applied to complications which did not always involve the brain.

Nor do I admit, like Musgrave and Sauvages, that hemiplegia occurring in an individual labouring under gout or rheumatism is on that account due in every case to apoplexy. Yet cases do occur in which, while the rheumatic attack goes on, transient hemiplegia shows itself which one cannot but refer to the rheumatism. I have, on a former occasion, related to you the history of a young girl who was admitted into the hospital, suffering from intense fever, excessive rachialgia, like that announcing variola, and from paraplegia. For three days,

I looked out for the eruption of variola; on the fourth day, I ordered her to be cupped, when all symptoms of paraplegia disappeared, but amaurosis and hemiplegia immediately set in. I then called the case an instance of *rheumatic hemiplegia* or of *cerebral rheumatism.* A few leeches were applied behind the ears, and two days afterwards pain was complained of in some of the joints, upon which the amaurosis and hemiplegia disappeared. For such cases of transient hemiplegia which, from the manner in which the paralysis alternates with other symptoms, is so manifestly due to the rheumatic diathesis, I admit the denomination of *rheumatic apoplexy.*

Hemiplegia may also arise from cerebral embolism in the course of rheumatism. One of the vegetations formed on the cardiac valves, as an event of endocarditis, is suddenly detached, and by blocking up a cerebral artery gives rise to sudden asphyxia of the brain by cutting off its supply of blood. If, now, collateral circulation makes up for the obstruction, the hemiplegia disappears, and the case is one of rheumatic apoplexy in the narrow sense of the word; but rheumatism is only indirectly at fault here, and the case is very different from the one in which rheumatism attacks directly the brain or its membranes, as it does the joints. I am, therefore, inclined to admit two varieties of rheumatic apoplexy, one dependent on congestion, and the other on embolism.

The rheumatic apoplexy of older authors, terminating in sudden death, remains still to be explained. In the majority of cases, the cause of death is not then to be found in the brain, but in the pericardium, the heart, or the large blood-vessels. Thus, pericarditis, when the effusion is rapid and considerable, may cause death by suddenly arresting the heart's action. Thus also, acute endocarditis may give rise to the same accidents, through organic or dynamic obstruction to the heart's action. Thus, lastly, a coagulum may form in the cardiac veins, in the right heart or the pulmonary artery, as a result of that remarkable tendency to spontaneous coagulation which the blood evinces in rheumatism, and this thrombosis will bring on asphyxia, which will carry off the patient rapidly, if not suddenly, in a state of stupor which may be erroneously regarded as cerebral.

Facts, ascertained within recent years, allow us, therefore, considerably to restrict the number of cases of rheumatic apoplexy; and we can scarcely say that the patient at No. 16 died from this cause. He complained, it is true, of mistiness of vision, and for a quarter of an hour, he was violently delirious; but I do not see that there was in his case the *apoplectic stroke proper.*

In other instances, the symptoms are, to a certain degree,

those of apoplexy, as, for example, in the case of the woman lying at No. 2. You remember how she felt irresistibly drowsy, and how she sat down in a dark corner of the kitchen, and how afterwards she dropped off to sleep while talking to me; you may recollect also that, for forty-eight hours, she suffered from aphasia. Such a case, with sudden well-marked cerebral symptoms, simulating those of hæmorrhage or congestion, is, I believe, an instance of apoplectic cerebral rheumatism. I will only mention, incidentally, à propos of rheumatic apoplexy, cases of profound stupor following on delirium in the course of an attack of acute articular rheumatism; or, again, those instances in which eclampsia comes on suddenly, and is succeeded by the usual stupor. But I will dwell more on cases like that of the young girl who had spinal pain and paraplegia, hemiplegia, and amaurosis, and at last pains in the joints. It is clear that, in her case, the rheumatism, whatever be the notion formed of its nature, attacked successively the spinal cord, the brain, and the articulations, probably affecting, in each region, similar anatomical tissues, but giving rise, at each spot, to very different symptoms. The lesions were too transient, however, to admit of their being ascribed to apoplexy, in the sense of Musgrave and Sauvages.

In one of our hospital nurses here, Seraphine, we have had another instance of these rapid fluctuations of functional disturbances. She has not menstruated for some time past, as she has now reached the period of life in which that function generally ceases. Three years ago her left wrist-joint was attacked with rheumatism, which caused marked swelling of the joint, and redness of the skin. She was then suddenly seized with vertigo and a sensation of weight in the head, and her limbs became so *paralysed* that she was unable to work. The pain in the head and nucha diminished, and her upper limbs regained some power, but the lower ones were still extremely weak, while she had a fixed and acute pain in the lower part of the spine. She was chiefly treated by veratria, and afterwards by spirits of turpentine. Her complaint resisted treatment for a long time; on several occasions she vainly tried her strength, and it was only after the lapse of *fifteen months* that she was enabled to resume work. She has enjoyed pretty good health since then, although her lower limbs have felt rather weak, and she has occasionally suffered from headache and numbness of the limbs.

During the summer of 1859, her finger-joints were painful and swollen; and her right foot, which was also swollen at the time, continued so until fresh symptoms showed themselves.

On January 11, 1860, during the night, she complained of violent cephalalgia and of pain in the right shoulder; the head-

ache diminished, but was succeeded by acute pain in the lower part of the spine, and numbness of the lower limbs; the swelling of the foot had disappeared. Eight days afterwards, the same symptoms returned. She complained of violent pains in the head and of mistiness of vision, of loss of power and numbness of the arms, chiefly the right; the pains next settled in the back, and the lower extremities became chiefly paralysed.

From January 20 to January 31, she was cupped along the spine on three separate occasions, and took turpentine capsules. Her symptoms improved markedly under the influence of this treatment; she had occasional headache, but it was never so violent as in the beginning; the pains in the back were also less intense, but the legs still continued to be very weak.

On February 7, the lower limbs were stronger, and the pain in the back less; the hands, on the contrary, were more painful, while pain was complained of in the back of the neck and in the head.

On the 8th, the legs were less numb, and felt stronger; but the hands, especially the right one, were numb. Eight turpentine capsules were ordered.

On the 10th, the patient felt stronger, and the numbness of the limbs was less.

On the 16th, the improvement was more marked, and the patient could darn. The turpentine was repeated.

On the 23rd, there was no pain at all down the spine, but slight pain was felt in the joints.

On March 1, the patient resumed work.

On the 8th, at twelve o'clock in the day, she was seized with violent rigors, and, after a few twinges in the arms, with a very acute cephalalgia, accompanied by a distressing sensation of intracranial pulsation. Some pain was also felt along the spine.

On the 9th, the headache had diminished, but the pain down the spine was worse. I prescribed two pills, containing one-fifth of a grain of veratria each.

The patient complained of weakness still on the following days. The same treatment was persevered in.

On the 29th, the right leg was still weak, while for the first time the left elbow became painful. The veratria was repeated.

On the 30th, pain in the elbow less.

On April 1, right foot swollen; left foot sometimes swells also, but less frequently. Veratria pills again repeated.

On the 16th, the patient felt well enough to begin work again. For a few days previously, her menses had returned. All swelling of the feet had disappeared.

In this case, gentlemen, you see rheumatic arthritis precede

for a few years the symptoms of paralysis, and thus account for their production. Could any doubt remain, an analysis of the phenomena would remove it at once, for the symptoms of paralysis and those of articular rheumatism proper will be seen to alternate in the most significant manner. At one time cephalalgia and sensorial disturbances are present; at another, spinal pain and weakness of the lower limbs; sometimes, again, the cerebral and spinal symptoms are replaced by painful swelling of the joints.

This woman, then, had alternately cerebral and spinal rheumatism; and the cerebral symptoms which she exhibited were of an apoplectiform character.

There is just now in one of my wards a poor woman of the name of Marie, whose very stout appearance would never lead one to suspect that she may labour under a nervous affection. And indeed the symptoms which she presents are not due to hysteria, but are evidently referable to the rheumatic diathesis. She first had an attack of acute articular rheumatism which seized on the wrists, and then attacked the head, producing stupor for a day or two. The spinal cord was next attacked, and paraplegia followed. For the space of four months, this poor woman thus presented mobile symptoms, suddenly shifting from one organ to another, from the brain to the spinal cord, and from the cord to some part of the limbs.

The other day you saw that she was utterly unable to walk, she felt giddy and stumbled; she had a vacant look, and her tongue was furred; she had great difficulty in collecting her thoughts and in expressing them in words; she looked as if she were drunk. To-day she has had mydriasis and imperfect vision, which set in suddenly. She occasionally suffers from exquisitely painful neuralgias, and I mentioned her case when on the subject of neuralgias. Her symptoms are sufficiently well marked, and their relation to rheumatism sufficiently clear to justify one in giving to them the name of *rheumatic apoplexy* of the brain and spinal cord.

This, in my opinion, is the apoplectic form of cerebral rheumatism, and it should not be ascribed, as Musgrave and Sauvages do, to the occurrence of effusion in the nervous centres.

I now pass on to the *delirious* form, which is more frequent than the apoplectic. The delirium has nothing peculiar about it in the majority of cases, and resembles that which occurs in other diseases, such as typhoid fever or variola, with this difference, however, that it generally terminates in death. This form usually runs an acute course. It lasts one, two, or three days, and is succeeded by stupor, the patient dying comatose, that is, with apoplectiform symptoms. In some cases, the delirium runs a slow course, and becomes truly chronic.

In 1861, I had under my care, at No. 7, in the male ward, a young man suffering from acute articular rheumatism, who for a whole month was delirious. Sometimes the delirium resembles puerperal mania, that form which lasts from eight to fifteen, thirty days and more, and either disappears spontaneously, or after the administration of a purgative or of bark. The interesting case recorded by my colleague, Dr. Mesnet, is an instance of this.[1] The subject of it was a young man, twenty-three years of age, who had just experienced heavy money losses, and who had been guilty of various excesses, and was therefore depressed in mind and weakened in body. He first complained of some vague pains in his joints, and then exhibited symptoms of pleurisy, which remained stationary; after this fresh pains set up in some large joints, the knees, the arms, and afterwards the ankles. These pains, which had come on suddenly, prevented all motion, and were accompanied by diffuse redness round the joints, without intra-articular effusion. No doubt existed as to the rheumatic nature of these symptoms. When the knees and shoulders became involved, the patient's mind got, as it were, benumbed; he lay in a state of hebetude, answered slowly questions put to him, had a difficulty in finding words and in collecting his ideas, and showed indifference to everything. A few days later, the relation between his cerebral condition and his articular pains became manifest; when the pains disappeared, the patient's intellect was more confused and acted more slowly; when they returned, he talked more. His prostration was soon succeeded by agitation, by violence, hallucinations of sight and hearing, illusions, delirious fancies; he believed himself to be the object of suspicions, of pursuit, and the victim of machinations, &c. A few days afterwards, in addition to his delirium, his movements became choreiform, he kept constantly flexing and extending his fingers, and could not raise his hand to his lips; he spoke in a curt interrupted manner, and swallowed with rapidity and with a kind of convulsion. The delirium, which had at first come on in paroxysms, became continuous as soon as the symptoms of chorea showed themselves. The patient, under the influence of hallucinations, was constantly trying to get up in order to avoid the evil-disposed persons by whom he fancied himself to be surrounded, or in order to run away from the importunate voices which annoyed him. Sulphate of quinine in gradually increasing doses was prescribed, and a remarkable improvement followed. The choreic movements, the agitation, the hallucinations, the delirious fancies, ceased, but the intellectual confusion continued for another fortnight. After that it disappeared of itself by

[1] Archives générales de Médecine, June 1856.

degrees; health and strength returned, and a complete cure was at last obtained after two months and a half.

I will return presently to this coincidence of rheumatism and chorea, but I have related this case in order to show you that the delirious form of cerebral rheumatism may present two very distinct varieties: 1st, an acute form, which is grave and ends fatally; 2nd, a chronic form, which is much less fearful. A third variety might be admitted, namely, that which is brought on by purulent arthritis; but there is not then cerebral rheumatism properly speaking. The delirium is a result of the suppuration, and is analogous to that which comes on in pyæmia or in diseases of low type. It begins with a slight disturbance of the intellect, and after a time the delirium becomes more marked and more continuous; there are muttering and carphology. Such a condition differs widely from that which characterises cerebral rheumatism.

I now come to the *meningitic* form. The name given to this variety is as bad as that of delirious or apoplectic, as I will presently show; but let us first enquire what its symptoms are. I need not remind you that the invasion of ordinary meningitis is generally announced by vomiting, pain in the head, which is sometimes fearful, by constipation, and in children by convulsions. Now, these symptoms never occur in the so-called meningitic form of cerebral rheumatism. Thus, vomiting is generally absent, and there is only delirium, which is remarkable on account of the suddenness with which it sets in, and which rapidly passes on to stupor. Such is the course run by the disease even when, after death, dissection shows the lesions which characterise meningitis. This form, then, as far as the symptoms are concerned, does not differ from the delirious, from which it cannot be distinguished during life. It is consequently an anatomical, not a clinical, form of the disease. In some rare instances, however, as in a case recorded by Dr. Marrotte, considerable effusion may take place rapidly, and symptoms may then show themselves indicative of compression of the brain, such as hebetude, dilatation of the pupils, and coma. The case is, then, one of true acute hydrocephalus.

I next pass on to the *choreic* form, which has not been sufficiently described by authors, but which deserves a special place by the side of the preceding varieties. I have already told you, when I spoke of chorea, that Dr. G. Sée[1] was the first[2] to ascribe

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[1] De la Chorée. Rapports du rhumatisme et des maladies du cœur, avec les affections nerveuses et convulsives. (Mémoires de l'Académie de Médecine, t. xx. 1850).

[2] [The following extract from an essay by Dr. Bright, "On Cases of Spasmodic Disease accompanying Affections of the Pericardium," in "Medico-Chir. Trans." vol. xxii. 1839, clearly shows that long before Dr. G. Sée

this complaint to a rheumatic diathesis, acting on the brain or the spinal cord. This view is based on authentic cases, but it should still be accepted with a certain amount of reserve, for, like all innovators, Dr. G. Sée has exaggerated the importance of these cases, and of the inferences which might be drawn from them; his exaggeration served to awaken more the attention of the profession.

Dr. Sée asserts that a child who has had one or several attacks of acute articular rheumatism will sooner or later have St. Vitus's dance. You know that rheumatism pretty frequently shows itself during the convalescence of scarlatina; now, chorea pretty often comes on after this rheumatism. Conversely, a child who has had one or several attacks of chorea will sooner or later have rheumatism. It even happens in some rare instances to find chorea come on during an attack of acute articular rheumatism. Recall to mind a case which I related to you in my conference on chorea, that of a girl who was caught round the body by a man on a dark staircase, and became shortly afterwards affected with unilateral chorea. This was soon replaced by acute articular rheumatism, and when the rheumatism got well, the chorea returned. But this is not all: experience has shown that chronic endocarditis is pretty frequent in choreic children, and that pericarditis is equallly so in children and adults who are subject to St. Vitus's dance. Now, as you are aware, endocarditis and pericarditis are both brought on by rheumatism.

Thus the relations of articular rheumatism to St. Vitus's dance are proved directly and indirectly.

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published his memoir, the connection between chorea and rheumatism was a well-known and accepted fact in this country.

"With regard to the connection between chorea and inflammation of the pericardium, when called upon the year before last to deliver the Lumleian Lectures at the College of Physicians, I took occasion to state that for some years I had been persuaded of the existence of such a combination, and little attention has hitherto, as far as I know, been paid to the subject, although the combination of this spasmodic disease has been long recognised. In the very excellent 'Syllabus or Outlines of Lectures on the Practice of Medicine,' published at Guy's Hospital, I find, in the edition of 1802, rheumatism distinctly stated as one of the existing causes of chorea: and in later editions, as in that of 1820, I find it stated that 'chorea sometimes alternates with rheumatism,' but through what organ or by what intervention this occurs is not conjectured."

Dr. Bright's opinion was that the *phrenic* nerve is inflamed in such cases, and that through it the irritation is conveyed to the spinal cord: hence the spasmodic seizures. Bouillaud (Maladies du Cœur, vol. i. p. 512) also ascribed to irritation of the respiratory nerves the nervous symptoms which occasionally supervene in the course of an attack of pericarditis; but this opinion does not tally with the fact made out by Dr. Kirkes, from an analysis of 36 cases of chorea, that this affection is oftener associated with endocardial than pericardial disease.—Ed.]

Lastly, cases of acute articular rheumatism do occur in the course of which chorea manifests itself. The relation between the two affections cannot then be doubted, and I may remind you of a case in illustration of this, which I have already related to you, that of a young girl, the daughter of a tailor in the rue Richelieu, whom I saw in consultation with Dr. Legroux. She had been suffering from acute articular rheumatism for the last ten days, and for two days previous to my visit, violent chorea had set in, with delirium, inability to eat or drink, and constant and violent vomiting. She died from the violence of the chorea. We can, therefore, infer from such cases that acute articular rheumatism is sometimes transformed into St. Vitus's dance, that is, into a cerebral affection, which is sometimes grave and sometimes mild. A choreic form of cerebral rheumatism should, therefore, be admitted in my opinion.

I now pass on to an important question, namely, what is the *nature* of cerebral rheumatism?

When one thinks of the facility with which rheumatism generally attacks and brings on inflammation of serous membranes, the first thought which occurs to the mind is that cerebral rheumatism is merely meningitis. We very frequently see rheumatism pass from the joints to the pericardium, or to the pleura, and less frequently to the peritoneum. Now, if one considers that the arachnoid membrane is identical with the pericardium and the pleura, there is no reason for refusing to admit that it may, like them, be affected in articular rheumatism. Hence, when great cerebral disturbances occur in the course of a rheumatic attack, one is tempted to say that there is arachnitis, just as one would say there is pleuritis if pulmonary symptoms arose. Reasoning is, therefore, in favour of those who maintain that rheumatic meningitis and pleurisy are similar. But let us examine whether anatomical experience confirms these theoretical views.

In the majority of post-mortem examinations, nothing has been found except occasionally some congestion of the pia-mater, as I found in the man who lay at No. 16. In most cases, I repeat, nothing at all is found, no fluid in the ventricles or in the arachnoid sac, no injection of the cerebral tissue.[1]

[1] [Sir T. Watson has recorded four cases of acute articular rheumatism, complicated with carditis and head-symptoms—the latter suggestive of inflammation of the membranes of the brain—which was disproved, however, by an examination of the body after death. In three of these cases, some serous fluid was found in the meshes of the pia-mater and in the lateral ventricles. (Lectures on Physic, 4th ed. vol. ii. pp. 303–309.)

A more remarkable case cannot be found than that related by Dr. Latham in his "Clinical Lectures:"—"One of the children of Christ's Hospital had, in the opinion of all who saw him, the severest inflammation of the brain. The attack was sudden, with great heat and frequency of pulse. He had delirium

As no material change can be detected, the advocates of meningitis appeal to the rheumatic nature of the meningitis, and to the fact that rheumatism is a complaint in which there is no tendency to suppuration. I admit that articular rheumatism does not show any tendency to form pus, or to leave fibrinous deposits inside joints. The advocates of meningitis, therefore, assert that, when rheumatism attacks the meninges, meningitis results, just as arthritis follows when the disease

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and convulsions, and pointed to his forehead as the seat of his pain. In three days he died, and, upon dissection, not a vestige of disease was found within the cranium; but the heart was exclusively the seat of the disease, and no other part of the body discovered the slighest morbid appearance. The disease of the heart was not confined to its investing membrane; it was the most intense inflammation, pervading the pericardium and the muscular substance." This case is detailed at full length by Mr. Stanley in vol. vii. of the "Medico-Chir. Trans." In a second instance, mentioned by Dr. Latham, "the whole force of the treatment was directed to the head, from a belief that the brain was inflamed. Upon dissection, the brain and its coverings were found in a perfectly healthy and natural state; and the pericardium, towards which during life there was no symptom to direct the slightest suspicion of disease, discovered the unequivocal marks of acute and recent inflammation."

In his essay, "On Spasmodic Diseases accompanying Affections of the Pericardium," published in "Medico-Chir. Trans." vol. xxii. Dr. Bright reports the case of a young man who had been suffering from acute rheumatism for six days, when spasmodic symptoms appeared, increased rapidly in severity, and were shortly accompanied by delirium. This ultimately became so violent that it was found necessary to put the unfortunate sufferer under restraint. He died at the expiration of three weeks; and on dissection, the brain was found perfectly healthy, and the pericardium and endocardium presented unequivocal signs of recent active inflammation.

Dr. G. Burrows, in his essay "On Disorders of the Cerebral Circulation," at p. 188, mentions the case of a shop-boy who died, after seven days' illness, in a state of restlessness and delirium. On dissection, the brain and its membranes were found normal, while the pericardium was found covered over with fresh lymph; and "upon the anterior surface of the left ventricle of the heart there was a white spot, about a quarter of an inch in diameter, which appeared to be formed by concrete pus."

In his admirable "Lectures on Delirium and Coma," delivered before the College of Physicians in 1850, Dr. Todd gives the case of a young woman who, after suffering for some days from rheumatic fever, was seized with delirium, and in a few hours afterwards had a convulsive fit, succeeded by coma and death; yet the closest examination of the parts after death, while it exposed extensive inflammation of the pericardium, could not detect a trace of inflammation of the brain, which, together with its membranes, was unusually pale.

In two cases of acute articular rheumatism, complicated, in the one case, with occasional delirium and choreic twitching of the voluntary muscles, for three days, and in the other, with occasional delirium and slight opisthotonos, for two days, mentioned by Dr. Fuller (On Rheumatism, Rheumatic Gout, &c. pp. 204, 206), an examination of the body, after death, disclosed no inflammation of the brain or its membranes.

Again, in a patient who died lately in the Middlesex Hospital, under the care of Dr. Murchison, of rheumatic fever, complicated with delirium and marked head-symptoms, no alteration of the brain or meninges was found after death.—Ed.]

seizes upon a joint; and, as in rheumatic arthritis, no fibrinous exudations occur, neither are these met with in rheumatic meningitis.

To this purely theoretical way of reasoning from analogy, I will oppose a practical argument also drawn from analogy.

The serous membranes of the pericardium and pleura are anatomically identical. Now, we daily meet with cases of rheumatism in which the pericardium and pleura are involved, but within a few hours of the occurrence of such complications physical signs reveal the existence of unquestionable organic lesions. Thus, on ausculting the lungs, the respiratory murmur is found to be less distinct, while on applying the stethoscope over the heart, a friction-sound, like the creaking of new leather, is heard. After the lapse of another twenty-four hours, all the physical signs of effusion, or indicating the presence of false membranes, are detected.

Now, since the arachnoid is anatomically identical with the pericardium and pleura, how, it may be asked, should it be privileged to escape the common law, and why should not fibrinous deposits and other exudations be found in its sac, as in that of the pericardium and pleura? But as no such changes are found in the bodies of individuals who have died of cerebral rheumatism, we are justified in coming to the conclusion that meningitis was not set up.

It would be vain to try and account for the absence of the lesions of meningitis by the rapidity with which death occurred, and to assert that the same thing would happen in the case of pericarditis and pleurisy if the patient were to die at the onset of these complications. This argument might apply to the case of my patient at No. 16, as he died very rapidly; but it cannot be applied to all the cases which have been observed. For this pretended arachnitis has been known to last from two to six days, and as no lesion was found in such cases, we are compelled to admit that there could have been no meningitis. In cerebral rheumatism, then, we find neither the symptoms nor the usual anatomical changes of inflammation of the meninges.[1]

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[1] [In some rare cases, however, it would appear that there may be true meningitis characterised by the formation of pus. Thus, in a female patient, who died under Sir T. Watson's care in the Middlesex Hospital, after symptoms of cerebral inflammation supervening upon acute rheumatism, unequivocal pus was found smeared over the hemispheres. (Lect. on Physic, vol. ii. p. 302.) In vol. xxix. of the *Medical Gazette*, the history of a similar case is related by Dr. Fyfe, of Newcastle. A man, 36 years of age, after suffering for some days from acute rheumatism, was seized with delirium and unequivocal symptoms of cerebral inflammation. Life continued for five days longer, and throughout that period there was either muttering delirium or a state of perfect coma. On the fifth day, at noon, he died; and dissection showed the

But what is it that happens in such cases, and what is my opinion of the nature of cerebral rheumatism, if not in all, at least in the majority of instances? Allow me to make a digression, in order to explain my meaning thoroughly.

When the brain and the spinal cord, or the peripheral nervous system, are concerned, one is, in general, satisfied with explanations which are by far too easy. Thus paralysis, which sets in suddenly, is ascribed to congestion or softening of the brain, or hæmorrhage into it. The existence of hæmorrhage and softening is frequently demonstrated; but it is not so with congestion, which is too easily admitted, and without any other reason than that there could have been no other lesion.

But let us analyse analogous cases. An individual becomes affected with chorea, which resists treatment for the space of four months, and which, as usually happens, is complicated with paralytic and convulsive phenomena. For, as you are aware, one half of the body is, in such cases, weaker than the other, so much so indeed that the dynamometer marks 1 for one side and 19 for the other. There is, at the same time, then, muscular paralysis, convulsion, and, frequently, even disturbance of peripheral sensibility, anæsthesia, or hyperæsthesia. The nervous system is, therefore, sufficiently disturbed to induce the belief that the spinal cord is disorganised, and even the brain itself, as there is often impairment of the intellect.

But if you examine the bodies of persons who have died from the violence of the chorea, and most carefully search in the brain and spinal cord, you will find neither intense congestion, nor softening, nor extravasation; in a word, you will detect no serious lesion which can adequately explain the symptoms noted during life. Up to the present time, at least, no such lesion has been detected.

The same thing happens in other neuroses, in tetanus, for example. An individual has undergone a trifling surgical

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membranes of the brain covered with lymph and pus, the vascularity of the brain enormously increased, and the lateral ventricles distended with serum.

Dr. Fuller, in his remarkable work on "Rheumatism," at p. 302, 3rd edit. mentions an analogous case, which came under his observation in St. George's Hospital. A man was admitted, under the care of Dr. Seymour, with his joints inflamed and swollen. One day his knees, which had been greatly swollen, became very much smaller and flaccid, and, coincidently with the subsidence of the swelling, he complained of pain in the head, became paralysed on one side, and expired in the course of thirty-six hours. On opening his body, a large quantity of greenish-looking purulent matter was found smeared over the greater part of the surface of the left hemisphere, and there was considerable effusion into the ventricles.

Dr. Fuller believes that in all such cases the cerebral inflammation does not appear to have been a *simple* extension of the disease, but to have been excited by the concentration of the rheumatic virus upon the brain in consequence of the sudden subsidence of articular inflammation. (Loco citato, p. 303.)—Ed.]

operation, say a month ago; he is nearly well, and there scarcely remains a few granulations which require to be touched with caustic before the cicatrisation is complete; when, suddenly, stiffness is complained of, first in the jaws, then in the neck, without any fever being lighted up, and is soon followed by the fearful convulsions and rigidity which characterise tetanus, and last from four to eight days, until death closes the painful scene.

Here is a very grave and powerful neurosis, affecting motility and impairing the intellect at the last, since death is preceded by stupor; and yet, on dissection, nothing is found, absolutely nothing.

Look at hydrophobia again. Nothing has been found after death to explain the phenomena of that fearful complaint. There is just now, in one of my wards, a woman suffering from tetany, that curious affection in which one or both hands are rigid, the fingers straightened, with their ends closely pressed together, and giving to the hand the appearance of a beggar's hand stretched out in the act of begging. This paroxysmal complaint is merely after all local tetanus, affecting the fore-arm and hand, and is unquestionably of nervous origin. But it does not depend on a definite lesion, such as inflammation of, or hæmorrhage into, the nervous centres; and although it may possibly be due to transient paroxysmal congestion, the fact can be more easily assumed than demonstrated. At all events the nervous system must be modified in some way or another in this complaint.

In an outbreak of amaurosis which suddenly attacked the inmates of the Fénélon Asylum, ophthalmoscopic examination of the eye detected nothing abnormal. Hemeralopia often occurs epidemically in large barracks, or on board ships, independently of any change in the hygienic circumstances of the individuals, and unattended with abnormal appearances in the eyes that are affected. It gets well almost spontaneously, disappearing in the same way as it came on, and leaving us as much in the dark as to the cause which produced it as to its anatomical constitution. Shall one say again that congestion was present? but such an explanation would be unsatisfactory.

In true meningitis, when disturbances of innervation exist, these are not due to inflammation of the cerebral meninges, but to the circumstance that the brain-substance itself is involved in the congestive or inflammatory process. We find after death anatomical proofs of this extension of the inflammation. But the case is very different with cerebral rheumatism, in the course of which true symptoms of meningitis do not show themselves, while after death no meningeal or cerebral lesions are discoverable. We are thus led to infer, both from clinical observation and from reasoning, that in consequence of the

cerebral rheumatism, the nerve-substance has probably undergone a modification analogous to that which is believed to occur in tetanus, hysteria, &c., a modification the nature of which is yet obscure and not anatomically demonstrable, but which nevertheless exists, as everything at least seems to indicate, although it cannot be referred to any nosological type.

From these considerations, to which I might add many others, I hold to the opinion that the phenomena of cerebral rheumatism are, in general, those of a neurosis, much more than of an inflammation or even a congestion having definite anatomical characters which can be easily made out.

I now pass on to the *mode of occurrence* of cerebral rheumatism. According to most writers on the subject, there must already be articular rheumatism before cerebral complications can arise; in other words, cerebral rheumatism cannot occur at the very first onset, or at least has never been known to precede the joint affection. It may be, however, that this happens more frequently than is believed, as facts will show. I had lately under my care a man who complained of a very intense pain along the spine, and who was paraplegic. I at first thought that he was going to have small-pox, but no eruption occurring at the usual time, I examined him more carefully, and suspected acute myelitis; but a few days afterwards he was seized with articular rheumatism. Previous to affecting the joints, the rheumatism had therefore attacked the spinal cord first.

I have already mentioned the case of a young girl who was admitted under my care, three or four months ago, with symptoms of threatening variola. At first she presented symptoms of spinal, and then of cerebral, disturbance, with amblyopia, but all these disappeared as soon as acute articular rheumatism showed itself.

In both these instances, then, spinal and cerebral lesions preceded the joint affection. Other cases of the kind may occur, as you may imagine, and it may happen that light is not thrown on them by the supervention of rheumatism in the joints, so that they may be mistaken for cerebral fever, instead of being recognised as cerebral rheumatism. Cases like mine justify one in admitting the possible occurrence of primary cerebral rheumatism, although the joint affection is more commonly the first to show itself. The same conclusion applies to the other manifestations of rheumatism, such as endocarditis, pericarditis, and pleurisy.

You probably recollect the case of a young man who was admitted under my care, with acute endocarditis, as shown by fever and a blowing murmur at the heart's apex. After a few days, the rheumatic nature of the complaint was proved by the

supervention of pain in the joints. The patient had never had rheumatism before, and it was clear that the disease had attacked the endocardium primarily, instead of secondarily, after the joints.

By the side of this case, I will relate another, in which actual proof was not obtained, as the rheumatic influence which caused endocarditis did not subsequently give rise to articular disease, just as it must have sometimes occurred that primary cerebral rheumatism has not been followed by arthritic manifestations. The subject of this second case was a young girl who had an attack of acute endocarditis, which ran its course without being corroborated by the supervention of articular rheumatism. But there is no reason why we should not say that the case was one of primary rheumatic endocarditis, not followed by articular rheumatism, just as we meet with instances of rheumatism without endocardial complication. Such cases are very rare, and in the immense majority of instances articular rheumatism precedes the cerebral, cardiac, or pleuritic manifestations.

I have told you that cerebral rheumatism was, in my opinion, a neurosis, and not a rheumatic inflammation; and that the cerebral centre could be attacked primarily before the joints, so that you may infer from those statements what my answer would be to the question whether cerebral rheumatism is due to *metastasis*. I must, in the first place, define what I mean, and what should be meant, by the term metastasis, and I will do so by giving you examples in illustration.

Acute articular rheumatism is an affection with multiple manifestations, which involves four, ten, thirty, and sometimes a hundred joints at the same time, as when it attacks simultaneously the articulations of the hand, foot, and vertebral column. Now, when the disease migrates from one knee to the other, we do not say that there is metastasis, but simply that the rheumatic influence which yesterday affected the right knee to-day involves the left knee, and will probably attack some other joint to-morrow. It is the same morbid cause which seizes on various articulations in succession, and affects parts with which it has a pathological relation. Such a case is not one of metastasis.

But I will now give you instances of true metastasis. An individual has the mumps, that strange epidemic complaint, which is in the highest degree contagious, and is characterised by sudden swelling of first one and then of the other parotid gland, the secretion of which is diminished or suppressed, and is accompanied by intense fever. All these symptoms disappear, after four, six, or eight days at the most, some diminution of the salivary secretion alone remaining. This disease is generally

of a mild character, but not always so, for it occasionally happens that the swelling of the glands goes down suddenly, and that the patient exhibits nervous symptoms which are sometimes extraordinary. He remains in that condition for a day or two, and then a testicle is suddenly affected in a man, or a mammary gland in a woman. This is an instance of true metastasis. The primary lesion disappears, while an organ essentially different from the first becomes the seat of disease. There is metastasis because there is no necessary relation between the primary morbid phenomenon and the testicle, none at least analogous to that which exists between rheumatism and the joints.

Now, in cases of cerebral rheumatism, is there metastasis? Certainly not, because the rheumatism does not leave the joints to seize upon the brain; it has spread, dispersed itself, I would be disposed to say, so as to involve a part which it had not previously attacked, but it still persists in the joints after it has affected the brain. Hence there is some ground for supposing that, in involving the brain or the meninges, the rheumatism has merely selected a fresh seat, in the same way as when it spreads to the pericardium or the pleura. It is true that, when the pleura is seriously inflamed over a large area, the rheumatism leaves the joint after a few days, but, I repeat, not through metastasis, but in accordance with the law laid down by Hippocrates: Duobus laboribus simul obortis, non in eodem loco, vehementior obscurat alterum.

In the case of the man at No. 16, the articular pains were present a few hours before the cerebral complication arose; they had become less severe under the influence of quinine, but they did not disappear suddenly, as they do in real metastasis. As to the woman at No. 2, she exhibited cerebral symptoms during the continuance of the pain, and after those symptoms had disappeared, the joints continued to be painful.

One may often be misled into believing that the rheumatism has left the joints when it attacks the brain, from the severity of the cerebral symptoms masking that of the joint-affection. The patient, in his delirium, tosses about wildly, moving in every direction the limbs which he previously kept motionless on account of the pain in his joints; and because he is no longer conscious of this pain, those about him believe that his joints are no longer affected. But this is evidently a mistake; the articular rheumatism still persists, for there are still swelling and redness, and exquisite sensibility, but the latter is masked by the delirium, and by the different nervous condition in which the patient is. Under whatever aspect we view the question, therefore, either in the light of general pathology or in that of

the phenomena observed in the course of an attack of cerebral rheumatism, we are justified in not regarding the implication of the brain in rheumatism as due to metastasis.

Let us now enquire into the possible *causes* of cerebral rheumatism, and, first, into those that are independent of the peculiar treatment employed, for certain modes of treatment have been accused of favouring cerebral complications in rheumatism. When I related to you the case of the woman at No. 2, who had almost simultaneously articular and cerebral rheumatism, I asked you to bear well in mind the fact that in 1848, after violent emotions, she had manifested symptoms of brain-disease, and had been treated for insanity at the Salpêtrière for thirteen months. This woman then showed an unquestionable predisposition to brain-disease (whether this was mania, epilepsy, or lipomania, it matters little). Subsequently, on becoming affected with articular rheumatism, before the disease has spread much, she is seized with cerebral rheumatism, which gives rise to stupor of forty-eight hours' duration. The patient got well nevertheless.

On the other hand, the man at No. 16 was addicted to drink; he was in a state of constant excitement, and was, as it were, stupefied by spirituous liquors, so that his brain was predisposed by this permanent irritation to get disordered. On his being affected with acute articular rheumatism, his brain gets soon implicated, and he dies.

In 1825, I attended, in St. Martin Street, a merchant suffering from acute articular rheumatism, who exhibited such severe brain-symptoms that I told his friends in the most positive manner that he would not get well. I was, in consequence, requested to cease attendance; but after a few days the patient's fierce delirium disappeared, and he recovered perfectly. Some time afterwards he had a second attack of acute articular rheumatism, attended with excessive pain, considerable swelling, livid discoloration of the integuments, and I detected gaseous crepitation, deep inside some joints, indicating an incipient stage of gangrene. This time the patient died.

Now, all the brothers and sisters of this man had been, or were, insane, and it was a matter of surprise with those who knew his family that he was the only member of it who had not yet become insane. On his getting rheumatism, however, he is seized with cerebral rheumatism, in consequence of his hereditary predisposition to diseases of the brain. I have, in another lecture, mentioned the case of a woman, many members of whose family were insane, and who, on falling ill of rheumatism, was carried off by cerebral rheumatism.

In cases, therefore, of cerebral rheumatism, we learn, from

the previous history of the patient, that he has at some period or other shown grave cerebral symptoms, or that there exists in his family an hereditary predisposition to grave neuroses.

The same thing may occur in other diseases besides rheumatism. Thus, in individuals in whose family history neuroses or insanity may be traced, or whose brain is constantly stimulated by the use of spirituous liquors, fearful cerebral symptoms may develop themselves in the course of an attack of variola (as you very recently saw an instance in one of my wards), or after some severe injury. There is, therefore, an hereditary or acquired nervous predisposition, in virtue of which some individuals are liable to cerebral complications during the course of various affections, and especially to cerebral rheumatism in the course of an attack of articular rheumatism. Let us next enquire whether, as some practitioners believe, any particular mode of treatment favours the occurrence of cerebral rheumatism. There are, as you know, two chief and opposite methods of treating articular rheumatism. Some practitioners advocate *bleeding*, others administer *quinine*. The former ascribe to quinine the production of cerebral rheumatism; the latter, in their turn, accuse the practice of bleeding of bringing on that complication. In this discussion, which has been carried on without due regard to truth, and, occasionally, to rules of good breeding, the advocates of the quinine treatment have, apparently, come off worse, and for this reason:—

Few practitioners nowadays open a vein in acute articular rheumatism; of fifty physicians attached to the Paris hospitals, perhaps not more than four bleed, while the rest prescribe quinine. Now, say that each of them has ten cases of articular rheumatism under his care, there will then be 460 cases treated by quinine, and only 40 by bleeding. As a matter of course, the proportion of cerebral rheumatism will be much greater in the first than in the second group of cases, in fact, in the ratio of 460 to 40. But the conclusion has been drawn that a greater number of cases of cerebral rheumatism occur in the practice of those who give quinine. This is true if mere numbers be taken; but it is false if the numbers be compared in their logical proportion.

Dr. Beau and Dr. Briquet, who give quinine, affirm that it is a most dangerous practice to bleed in acute articular rheumatism, and they ground their opinion on the specious fact that repeated bleedings increase the amount of fibrin in proportion to that of the other constituents of the blood, and, therefore, favour the tendency to inflammation of the brain chiefly, which is weakened by the anæmia induced. On the other hand, the advocates of bleeding assert that, by exciting the brain, quinine renders it liable to the rheumatic influence.

Truth is mixed with error in these statements. More than sixty cases of cerebral rheumatism are now on record, seven or eight of which proved fatal after a course of bleeding, practised, as Dr. Briquet expresses it, in the orthodox manner. In other cases, which proved fatal, the patient was bled two or three times only, and was cupped several times. Of those who died in consequence or in spite of the sulphate of quinine, some had taken a small quantity of the drug, others moderate, and others, again, a very large amount of the salt.

The woman at No. 2 had not been bled, and had not taken quinine; she was nevertheless attacked with cerebral rheumatism, but got well.

The man at No. 16 took, for four days, a scruple; for three days, thirty grains; and on the last day of his life, forty grains of quinine; and these moderate doses did not bring on tinnitus aurium, or mistiness of vision.

In cases reported by Drs. Bourdon, Requin, and Gubler, cerebral rheumatism came on when the patient had only taken ten grains of quinine. Dr. Beau quotes a case in which, in spite of the cerebral rheumatism, he persisted in the administration of quinine, and the patient recovered.

To sum up, then, cerebral rheumatism does not seem to be brought on by any treatment in particular; it depends on the existence of a special predisposition, acquired or hereditary, which I have endeavoured to bring out prominently, and is not caused by bleeding or by quinine.[1]

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[1] [That the occurrence of head-symptoms in rheumatic fever is not due to blood-letting or to quinine is evident from the fact that such complications do occasionally arise in this country where the administration of alkalis or of calomel and opium have been the two favourite methods of treatment. When quinine has been given, it has generally been in combination with alkalis, and, at all events, never in the enormous doses which French practitioners are in the habit of using in rheumatic fever. In Dr. Fuller's opinion (loco citato, p. 205), we may expect the occurrence of cerebro-spinal symptoms, in the course of an attack of rheumatic fever, "not necessarily in those persons whose articular inflammations are most numerous and severe, but in those who are pale, weakly, and unhealthy; who have been much reduced by blood-letting, or by excessive and long-continued perspiration; who are attacked after over-long lactation, or during recovery from serious illness; or who, again, as Dr. Watson has remarked, experience a relapse after a long and tedious attack of rheumatism." Sir T. Watson and Dr. G. Burrows have both suggested that the head-symptoms may arise in consequence of a disturbance of the cerebral circulation, occasioned by the embarrassment of the heart's action, which results from the access of cardiac inflammation. But cases have been recorded in which no cardiac inflammation was present, and in which cerebro-spinal symptoms have nevertheless shown themselves. Dr. Todd mentions a few such in his already quoted "Lumleian Lectures;" and Dr. Fuller states that he has "seen eight cases in which slight wandering or delirium has arisen, and has continued for several hours, although the stethoscope has failed in detecting the slightest mischief within the chest, and the general symptoms have been inconsistent with the occurrence of inflammatory action within the cranium."

Now, as to the *treatment* of cerebral rheumatism? The cases should be divided into two groups: those in which the disease is merely imminent, and those in which it has broken out distinctly. Thus, when an individual, suffering from articular rheumatism, shows signs of commencing nervous excitement, becomes garrulous, complains of feeling hot all over, and speaks despairingly of his own case, cerebral rheumatism may be diagnosed as imminent; but can we do anything for the patient? I believe that the best plan consists in encouraging the articular manifestations of the disease. If they have abated, attempts should be made to bring them back by means of sinapisms, or of blisters to the joints. Opium and musk should, at the same time, be given internally. This, again, is, in my opinion, the most rational line of practice to pursue when the brain has become involved. I have succeeded in curing three patients who were under my care from the onset of the cerebral rheumatism by means of musk and opium. I have failed in other instances, and twice recovery took place without any active treatment having been had recourse to; one of these two last cases was that of the woman at No. 2, which is an illustration of the *vis medicatrix naturæ*.[1]

In his "Lectures, chiefly Clinical," at p. 140, Dr. Chambers relates the case of a letter-carrier, aged 24, who, from the very beginning of an attack of acute articular rheumatism, became violently delirious. He remained so for five days after admission. The joints were swollen and red during the delirium, and the redness and swelling disappeared simultaneously with the delirium. There was nothing in the man's antecedents to account for the delirium. He had no pericarditis, no endocarditis, no pneumonia. He recovered under the influence of a treatment by iodide of potassium in large doses, ammonia and bark, wine and beef-tea every two hours.

The presence of the rheumatic poison circulating in the blood is sufficient to account for the occurrence of cerebro-spinal symptoms; and in his admirable "Lumleian Lectures on Delirium and Coma," published in the *Lancet* for 1852, Dr. Todd has adduced full and copious proofs in support of the important fact that, although delirium, convulsions, and coma may result from cerebro-spinal inflammation, yet an altered relation of the circulating fluid is equally, if not more, energetic in their production. Now, the reason why such symptoms do not arise in every case of rheumatic fever seems to be that the rheumatic poison can only play the part of the lighted match, and that there must be, in addition, combustible material present. That combustible material is the nervous susceptibility of the patient, his peculiar predisposition to the development of cerebro-spinal symptoms, from acquired or hereditary causes, as so well pointed out by Professor Trousseau.—Ed.]

[1] [Dr. Fuller also recommends the combined administration of diffusible stimulants and opiates in these cases, provided the disturbance of the nervous centres be ascertained to be functional only, and unconnected with any cardiac, pulmonary, cerebral, or spinal inflammation. "Even when such inflammation exists, depletion should be seldom practised to any great extent, and we should rather endeavour to support our patient, whilst aiming at the relief of the more urgent symptoms, by means of blisters, mercurials, diuretics, and opium. . . . One exception only exists to the full though cautious exhibition of opium, namely, when there is a tendency to the supervention of coma. In such cases

opium is not only useless, it is decidedly prejudicial to the safety of the patient, who requires a more than usual amount of support and stimulus."

We have already quoted a case of Dr. Chambers', in which recovery took place after the occurrence of the most violent delirium in the course of rheumatic fever; and Dr. Fuller has related at full length the history of a girl, aged 19, Harriet Keating, who recovered after having been furiously delirious, and had violent tetanic spasms, whilst suffering from rheumatic fever complicated with carditis. Dr. Fuller mentions, also, that an equally favourable issue occurred in another case, which came under the care of Dr. Edward Dewees at the Coventry and Warwickshire Hospital. These instances of a successful termination of cerebral rheumatism may be added to those mentioned by Professor Trousseau, and show that the prognosis need not be necessarily fatal in such cases.—Ed.]

LECTURE XIX.

EXOPHTHALMIC GOÎTRE, OR GRAVES'S DISEASE.

The chief symptoms of the disease are three in number: Hypertrophy of the Thyroid Gland, Exophthalmos, and Palpitation.—The disease may be incompletely developed.—Nervous phenomena frequent.—Nature of the affection.—It is probably a neurosis of the sympathetic.—Cases and arguments in favour of this view.—Good results of Hydropathy.

Gentlemen,—You may have noticed at No. 34, in St. Bernard ward, a young woman who has a somewhat strange physiognomy. Her face has a savage expression, her eye-balls are prominent, and her complexion pale. She complains of palpitation of the heart; her pulse at the wrist is frequent, regular, and of normal volume and resistance. Her breathing seems to be impeded, and you could see that her thyroid gland was considerably hypertrophied. The coexistence of these three pathological phenomena—palpitation, hypertrophy of the thyroid gland, and prominence of the eye-balls—constitutes a morbid entity of which you will find numerous instances on record, and which has been designated under the names exophthalmic goître, exophthalmic cachexia, cachectic exophthalmus, Basedow's disease, &c.

Although ophthalmologists like Demours, Mackenzie, Sichel, and Desmarres, had already mentioned this complaint, which is so remarkable from its three prominent symptoms, Graves was the one who called attention to it, and afterwards, from Basedow giving a fuller description of it, it was known after his name. Hence, Dr. Hirsch, who perhaps did not know Graves's researches on the subject, has of late years claimed for this complaint the designation of *Basedow's disease.*

In some clinical lectures which I delivered in November 1860, I mentioned to you, on the authority of Stokes, that the credit of priority belonged to Graves in a great measure; and those of you who may wish for more proofs need only refer to the "Lectures on Clinical Medicine," published by the illustrious Dublin professor, and the chapter on *Exophthalmic Goître* in Dr. Stokes's work on "Diseases of the Heart." I leave to the professor of systematic medicine the task of giving you an historical notice of exophthalmic goître, and of doing justice to the authors who were the first to investigate the subject. As to myself, I mean

in this lecture to give you the clinical history of this singular and interesting complaint, to relate a few instances of it, so as to show it to you under different aspects, and to discuss its nature by the light of cases of the disease, which have now been published in pretty large number, and the value of which may be easily tested by fresh observations. I mean particularly to draw your attention to the treatment which has seemed to me the best, which is indicated by the very nature of the complaint, the only serious basis of all treatment, when we have not specifics at command, or cannot have recourse to methods approved by empiricism.

Many individuals suffering from exophthalmic goître will come and consult you on account of palpitation, but you will be at once struck with their strange look and their prominent eyes. The prominence of both eye-balls should immediately point to your diagnosis. On enquiry, you will find that the exophthalmos is of old date, that it increased by degrees, but that it sometimes does so to such a degree that the patient is afraid lest his eyes should fall out; he has a sensation as if these organs were going to drop out of their sockets, and experiences a difficulty in closing his eyelids completely, and his eye-balls are often partially uncovered during sleep. In a young woman at Clermont, whose case was published by Dr. Pain, the eye-balls were pushed forward so much that one of them actually came out of the orbit, and had to be put back with the fingers.

The exophthalmos is most marked under the influence of mental emotion, and at the menstrual periods. The coats of the eye generally present no alteration, and I have never noticed ulceration of the cornea.

When there is considerable prominence of the eye-balls, the anterior insertions of the recti muscles can be easily seen, and the spot is remarkable for its great vascularity.

There is generally no disturbance of vision, although the patient may become either long or short-sighted; frequently, however, the eye retains to a great degree the power of adapting itself to distances. I knew a man who could read at very variable distances, while the eye-ball and the pupil underwent certain modifications; thus, he had convergent strabismus and dilatation of the pupils when the object was held near his eyes, while the eyes recovered their normal position and the pupils contracted when the object was at a distance. It could be seen that, according to the difference in the position of the object, adaptation cost an effort, for there was a greater secretion of tears, which first increased the brilliancy of the eyes, and then dropped on the lower lid. Two patients complained of weakness of sight, and of occasional muscæ volitantes. I have never met with diplopia.

The double exophthalmos and the temporary disturbances of vision naturally attracted the attention of oculists, and interesting ophthalmoscopic observations have therefore been published. A very good summary was published in the Danish language by Dr. Withuisen, of the appearances found on ophthalmoscopic examination of the eye in a case of exophthalmic cachexia: "The ocular media were very transparent, and the retina was of a marked red colour from a bright injection of its vessels. The point of entry of the optic nerve was of a yellowish red tint, perfectly different from its normal one; the branches of the arteria centralis retinæ were more largely developed than usual, but did not pulsate. On each side of the optic papilla pigment was deposited in semilunar masses almost black in hue. These masses had a concave and distinct edge on the side turned to the papilla, but their other margin was convex and toothed. In both eyes, these masses were of larger size on the outer side."

This case is of considerable interest, and this is increased by the fact that an ophthalmoscopic examination was made during convalescence. It was then found that the hyperæmia of the fundus oculi was less, in fact, that the congestive appearances previously noted had nearly disappeared, but the masses of pigment had undergone no change. Other observers have also noted congestion of the retinal vessels without alteration of the ocular media.

To give this case of Dr. Withuisen its full value, I wish to state positively that it was observed with great care, and that it was an undoubted instance of Graves's disease, for "it had been ascertained, on making an external examination of the eye, that there were double exophthalmos and varicose dilatation of the vessels of the conjunctiva about the insertion of the recti muscles. The cornea was of normal convexity, but the pupil was dilated and the iris slow to contract. The anterior chamber was flatter than natural, probably in consequence of the prolapse of the iris. The patient had a somewhat strange look, expressive of surprise. She complained of being short-sighted, and of having a difficulty in looking steadfastly at the same object for some little time. There were occasional vertigo, and frequent pain in the eyes, with headache, and when she closed her eyes, she sometimes saw circles of fire."

I have quoted this case almost in full, because the eyes were examined with great care. It was thus made out that the membranes of the eye may be the seat of great injection, and of a modification of nutrition, giving rise to deposits of pigment and of a yellowish material on the retina. When I come to speak of the pathological anatomy of the disease, I will tell you the other alterations or modifications which dissection has shown,

and we shall then possess all necessary elements for discussing the mechanical cause of exophthalmos and the nature of the functional modifications of the organ of vision.

You must not think that there always is very considerable prolapsus of the eye-ball; in some cases, you must admit it on the authority of the people about the patient, unless you have known him yourself previously. Even when the eyes are not strikingly prominent, they have always a special look in them, and there is some transient or lasting disturbance of vision which indicates an abnormal condition of the eyes.

But if the prominence of the eye-balls may escape notice there are two other pathological facts which attract attention forcibly in nearly every case, namely, hypertrophy of the thyroid gland and palpitation of the heart.

The thyroid gland is sometimes very considerably enlarged; its two lobes may hypertrophy to an equal degree, but in the majority of cases, according to Graves, Stokes, and others, and according to my own experience also, the right lobe is the one chiefly affected. The transverse portion of the gland may be involved, so that a goître of considerable size is thus produced. The hypertrophy usually occurs gradually; it begins insidiously, as it were, and a casual circumstance alone reveals this condition to the patient. In a short time, the swelling of the thyroid enlarges more and more, simultaneously with an increase in the symptoms of the general disease. Occasionally there are periods of arrest, but the tumour has in some cases become sufficiently voluminous by this time to give rise to very appreciable modifications of the voice and of breathing. There is marked difficulty of breathing, especially when the patient lies on his back, either from the trachea being compressed by the weight of the tumour, or from its being enveloped in the hypertrophied portion of the gland which forms like a constricting band round it, as in cases of suffocating goître. In some instances, as I will show you presently, the hypertrophy takes place almost suddenly. Lastly, I will relate to you a case in which very striking hypertrophy was succeeded by atrophy, the gland becoming affected with true cirrhosis.

The changes in the voice which occur in exophthalmic goître may be due to the modifications of respiration, or to pressure on one of or both the recurrent laryngeal nerves. The voice may then be weak or hoarse. I need not observe that by modifying the contractility of the muscles of the glottis, pressure on the recurrent laryngeal nerves may have a share in causing difficulty of breathing.

Pathological anatomy clearly demonstrates in such cases that there is glandular hypertrophy proper, that is to say, hypertrophy of the glandular elements, of the *acini*; while, in

addition to functional changes which are the result of this condition, great development of the blood-vessels of the gland may be made out clinically. For on applying the hand over the swelling, an expansive movement may be felt, which indicates that there is something more than dilatation of the superficial vessels only. This expansive movement is sometimes considerably marked over the right lobe, and in a case mentioned by Graves, the swelling formed by the thyroid was mistaken for an aneurism. Dr. Vidal, of the Paris hospitals, has told me of another case in which a similar mistake was made. In both these instances, the mistake was found out before any surgical interference. But this might be decided on, unless the general symptoms of the disease be taken into account, for in exophthalmic goître, simple or double bellows-murmurs, with accentuation of the diastole, may be heard with the stethoscope, as in simple or cirsoid aneurism.

Everyone is agreed on these two facts: double exophthalmos and hypertrophy of the thyroid gland. When I come to discuss the nature of the disease, I will investigate their course and the paroxysms which they present, and will tell you how to interpret them. Meanwhile I pass on to the other great clinical fact which, with the two preceding, constitutes the symptomatic trio or tripod characterising Graves's disease: I mean, the state of the heart.

The patients complain of palpitation long before the exophthalmos and goître have attracted their notice, or that of their friends. There is violent beating of the heart, which, by pushing forward with force the generally emaciated chest-wall, soon produces prominence of the præcordial region, while the heart's impulse against the chest is so powerful that it can sometimes be heard from a distance. These pulsations increase in frequency and strength under the influence of mental emotion or of exertion of any kind, so that the patient cannot take any continued exercise. The valvular sounds are exaggerated, and are generally accompanied by a soft systolic bellows-murmur, audible in the large arteries also. The carotids pulsate more forcibly than natural, and they, as well as the jugular veins, have a share in the production of the sounds heard over the enlarged thyroid.

When we look at the group of symptoms made out by examining the heart, namely, violent but regular impulse and bellows-murmur at the base, we can easily understand how Stokes has been led to describe a variety of exophthalmic goître with hypertrophy of the heart. I must add that valvular disease was present in some of Stokes's cases, although he already saw that this was not the rule, and he therefore described separately exophthalmic cachexia complicated with organic disease of the heart when treating of the disease. This clinical division should,

I think, be retained, because, although exophthalmic goître is not, in my opinion, necessarily attended with dilatation of the cavities or alteration of the valves of the heart, yet such lesions may coexist with it, and may perhaps have been instrumental in bringing it on. But there is a wide difference between this interpretation and the theory broached by Stokes, namely, that the disease is merely a cardiac neurosis, to which all the morbid phenomena are due. Nor can I concur with Dr. Aran,[1] who has stated that the heart was always enlarged in cases of exophthalmic goître.

It might be asked with regard to Dr. Aran's cases, be it said in passing, whether there was merely dilatation of the cavities or true hypertrophy of the walls of the organ. For he simply stated that there was increased cardiac dulness. The majority of the cases which have come under my own observation, and those which have been published by Dr. L. Legros and Professor Teissier,[2] do not tell in favour of Dr. Aran's views, but as assertions made by such a sagacious observer should be taken into consideration, I will enquire how the discrepancy has arisen.

Aran, after making out that there was extensive cardiac dulness, tried to support his opinion by arguments drawn from the character of the heart's impulse and the pulsation of the carotids. But I will observe that the heart may beat, and the large vessels pulsate, with violence without there being necessarily hypertrophy of the heart, and these symptoms may exist in chlorosis and hysteria. In such cases, these transient phenomena are merely the consequences of perfectly determinate nervous states.

But it is important to ascertain whether the increase in size of the heart be a constant fact. It is not so, I think, and it may therefore be asked how my opinion is to be reconciled with that of the observers who believe that there is such an increase in size.

It has been stated by some that, in addition to palpitation and violent impulse of the heart, the area of cardiac dulness is increased. From this it might be inferred that the organ had increased in size. But it is a remarkable circumstance that the pulse at the wrist was, in the majority of instances, found to be normal, while, if there had been cardiac hypertrophy, the radial arteries would have been full and vibrating like the carotids. Had the heart been, on the contrary, passively dilated, the radial arteries would have been weak and perhaps irregular, and, in some cases, jugular pulsation might have been noted. But no

[1] Bulletin de l'Académie Impériale de Médecine, Paris, 1860, t. xxvi. p. 122, and following.

[2] Du Goitre exophthalmique, 1863.

such symptoms were ever found to be present, and the exaggerated beating of the heart and the increased area of cardiac dulness were the only ones in favour of the idea of cardiac hypertrophy. I have already suggested an explanation of the exaggerated impulse; and as to the other phenomenon, increased area of cardiac dulness, it is often present, but is merely apparent. For there are two kinds of præcordial dulness: one of which is absolute, and measures normally from 4 to 5 square centimetres (about 2 square inches), and the other relative, that is to say, dulness extending beyond the limits of the preceding, which may vary indefinitely, according to the relations of the heart to the lungs, according to the amount of obstruction to the circulation, and to the greater or less degree of thickness and density of the thoracic parietes. The relative dulness may measure from 10 to 12, 13, 14 centimetres transversely (from 4 to nearly 6 inches), and from 8 to 10 or 12 centimetres ($3\frac{1}{2}$ to 4 or $4\frac{1}{2}$ inches) vertically. This dulness may be readily made out in Graves's disease, because it frequently happens that the patient's chest-walls are very thin.

I lay great stress on this cause of error, because in a case of exophthalmic goître, in St. Bernard ward, several observers differed as to the presence or absence of cardiac hypertrophy, some believing that there was hypertrophy, on account of the increase in the area of cardiac dulness. Professor Bouillaud kindly examined the patient, and this eminent practitioner, whose experience in the diagnosis of heart-disease is so great, stated positively that there was no increase in the real absolute dulness, and that there was no hypertrophy of the heart.

The greatest care should be taken to determine the limits of the real or absolute, and of the relative, dulness, as the former alone is to be regarded as indicating hypertrophy of the heart.

Although there may not be active hypertrophy of the central organ of circulation, there may sometimes be dilatation of its cavities, for one may conceive that during the paroxysms of Graves's disease, when the heart gets fatigued after acting tumultuously, its walls, especially those of the right auricle, should get distended. At such times, percussion will show an increase in the area of dulness, but only temporarily.

The rule, therefore, is that Graves's disease is not necessarily attended with disease of the heart, but it may attack individuals who become subsequently subject to cardiac affections. The patient's previous history and the presence of signs indicating organic lesions will enable the practitioner to ascribe to each disease its proper share in the production of the cardiac condition.

From the cases of Graves's disease which I have examined, I

have come to the conclusion that this complaint does not necessarily bring on hypertrophy of the heart, but I can conceive that it may give rise to a lesion analogous to the cardiac hypertrophy which occurs during pregnancy, a condition which may be transient only, disappearing a few weeks or months after the primary complaint has been cured, or may remain permanently, as in some instances, which have as yet been rare. It seems, indeed, to be a well established fact now that the heart and other muscular organs may increase in size pretty considerably without being diseased on that account. Cases published by Dr. Larcher, the subjects of which were pregnant women, and the results of dissections made by Dr. Blot of the bodies of women who had died shortly after delivery, leave no doubt as to the physiological hypertrophy of the heart during pregnancy. Dr. Beau is himself of opinion that, if there be cardiac hypertrophy in Graves's disease, the lesion is curable.

There can be no question as to the enlargement of the blood-vessels in the neck. The carotid and thyroid arteries undergo important modifications; the latter especially increase markedly in calibre, both the trunks and their ramifications; the thyroid veins dilate in the same manner; and the blood-vessels are so largely developed that it would be an act of rashness to cut with a knife into the swelling formed by the thyroid gland. There is an increase in the amount of blood-vessels, and dissection proves that the vascular as well as the glandular elements of the thyroid are hypertrophied. The thyroid may be seen to expand, and on auscultation, a bellows-sound may be heard over it. Similar murmurs and pulsations may be observed over the region of the cœliac axis. These phenomena do not extend into the lower portion of the abdominal aorta, nor into the iliac and femoral arteries, and it is a remarkable circumstance that, while the heart and the cervical blood-vessels seem to indicate exaggerated activity and force of the circulation, the pulse at the wrist is not abnormally full. This clinical fact did not escape Graves, Stokes, Hirsch, and all those who have studied exophthalmic goître. It seems to indicate that the pulsations of the heart and of the cervical blood-vessels are due to some special cause limiting its action to the walls of those organs.

We shall see by and by what inference can be drawn from this localisation of the morbid action, with regard to the nature of the disease, by putting together pathological facts and the physiological phenomena which Professor Claude Bernard has so well studied experimentally.

As yet I have only spoken of the three principal symptoms which together constitute Graves's disease, but there are other secondary symptoms which should be taken into account. In some cases, there is diminished or capricious appetite; in others,

on the contrary, the patient is not easily satisfied, digestion is good, and yet there is progressive emaciation, and the patient's colour goes. Sometimes diarrhœa comes on, which increases the tendency to emaciation. When the complaint seems to improve, however, the diarrhœa diminishes and stops: the patient takes advantage of her voracious appetite, and recovers the appearances of good health. If she has not yet attained her full growth, she is then noticed to grow with prodigious rapidity, and to gain strength in a sustained manner. As fresh paroxysms return, these advantageous results of a good nutrition may disappear, but, as a rule, the paroxysms become less and less frequent from the time when nutrition begins to improve. Most of the women who have Graves's disease suffer from amenorrhœa also. In the beginning, menstruation is only disturbed, but it is after a time completely suppressed, and hopes of a favourable issue are not to be entertained until this function is perfectly re-established. This is an important prognostic sign. The amenorrhœa is accompanied by leucorrhœa, which is sometimes very profuse, and thus increases the tendency to debility.

The patient often also presents all the characters of anæmia, and, in some cases, even of well-marked chlorosis. The capricious appetite, the development of flatus in the intestines, the alternation of diarrhœa and constipation, the palpitation of the heart and the bellows-murmur heard over the blood-vessels, the extreme pallor of the face and of the mucous membranes, the œdema of the lower limbs, the disordered menstruation, and, in some cases, the marked changes in the patient's temper, might mislead one as to the nature of the affection, if all these phenomena were not secondary to, or concomitant with, the three great symptoms of Graves's disease. Several practitioners are inclined to ascribe to anæmia a share in the production of this complaint, but I am glad to be able to refer you to an essay, rich in cases, lately published by Professor Teissier, in which he has shown that exophthalmic goître may exist without anæmia. In four of his cases, there was not a trace of anæmia: the patients had, on the contrary, all the appearances of a fine and sanguine temperament; they were stout, had powerful muscles, and, moreover, had a sense of their strength.

The patient's temper is so altered that the persons about her can scarcely put up with her irritability, her want of grateful feelings, and her exacting ways. I have known a young lady, who was usually of a sweet disposition, become disrespectful and quick-tempered, almost violent. Besides this change in the temper, there is sleeplessness, an unpleasant complication, which, if it lasts, throws the patient into a state of perfect despair; she cannot find an easy posture, but keeps shifting

about in bed, and longs for the return of day; she feels perfectly weary, and yet cannot rest for a moment.

Now, what is the mode of invasion, and what the course, of this strange complaint? in what order do the symptoms succeed one another?

Without any very determinate cause to account for it, generally in persons of a nervous temperament, a certain degree of irritability becomes noticeable; the temper is not so even as it was. Within a short time, the expression of the face, and that of the eyes in particular, is in keeping with the sudden burst of displeasure, and the transient fit of anger. It is a remarkable circumstance, however, that the eyes permanently retain a strange look; they are unusually lustrous, and look larger. The exophthalmos soon becomes manifest, and then presents the characters which I have already mentioned. The patient is conscious of the mobility of her temper, and says that she often tries, but in vain, to repress it. She feels sad, and cannot account for her painful sensations; she complains of a sense of beating in the head, inside the eye-balls and along the neck, and expresses alarm at her palpitation, on account of its frequency and violence.

A medical man is consulted on account of the capricious temper, the strange look about the eyes and the palpitation of the heart; and, until the time when Graves's disease came to be described as a morbid entity, serious mistakes were frequently made. Practitioners who did not know this complaint thought that the peculiar mental condition of the patient and her palpitation were merely curious nervous symptoms due to anæmia or chlorosis, or to painful or irregular menstruation.

The patient, however, especially if a woman, drew the practitioner's attention to the swelling in her throat, and mentioned that for some time past she had had a sensation of fulness and of pulsation in that region; her statement was noted down, but was not taken into account, as at the outset the thyroid gland is not enlarged to a considerable extent. By degrees, however, as the throat swelled, the practitioner's attention was forcibly drawn to the coincidence of those three symptoms, palpitation of the heart, exophthalmos, and hypertrophy of the thyroid gland. This coincidence was looked upon as curious, and instances supposed to be analogous, in which it had been noticed, were recalled to mind, especially when the patient happened to be chlorotic. But as after all these three symptoms were rarely met with in combination, and were not detected at the onset of the complaint, the cases in which they occurred were regarded as curiosities, and the facts observed, remaining uninterpreted, were as a sealed letter.

Nowadays a mistake is less easily committed, and the

disease will be recognised whenever the simultaneous or closely successive development of the various phenomena which characterise it is observed. Do not think, however, that the diagnosis is always easy. A certain degree of care is necessary, in order to recognise these phenomena in the beginning, and the form which they assume, when they are in an incipient stage, should be well borne in mind. You should thus suspect this complaint when you find a lustrous appearance of the eyes coexisting with palpitation of a violence out of proportion with the organic condition of the heart, and you should banish all doubt when you find, in addition to the above symptoms, marked increase in the pulsation of the vessels of the neck, and slight hypertrophy of the thyroid gland. Bear in mind, however, that the latter symptom may be late in showing itself, especially if the patient be a male. This is not the case in women: hypertrophy of the thyroid is well marked in them when they consult a medical man, and it keeps pace with the increase in violence of the palpitation of the heart and the prominence of the eye-balls. In several cases, however, which have come under my notice, the goître was late in its appearance, although the patients were women.

Quite recently Dr. Cazalis, a distinguished physician of the Paris hospitals, did me the honour of consulting me about an engineer, thirty-five years of age, who presented all the symptoms of exophthalmic cachexia, and who complained of such violent palpitation that Dr. Cazalis and I paid particular attention to the organic condition of the heart. The most careful examination enabled us to say that there was no hypertrophy of that organ, for the transverse dulness measured two inches only, and no abnormal bruit was audible over the aortic and mitral orifices. The thyroid gland did not seem to be hypertrophied, but on carefully examining the anterior aspect of the throat while the patient was placed in a favourable posture, we made out that the throat was slightly larger on the right side. The increase of size was scarcely appreciable, but it was sufficient, when complemented by the other symptoms, to enable us to state positively that the patient was suffering from Graves's disease. I saw the patient four months after this, and then found slight swelling of the right lobe of the thyroid gland, with some aortic blowing murmur. He was, on the whole, considerably better.

Graves's disease is pretty common in women, but is relatively rare in men. Of fifty cases of this complaint collected by Withuisen, only eight occurred in males. For that reason, then, I will now relate to you the history of the patient whom I saw with Dr. Cazalis.

He consulted Dr. Cazalis, for the first time, on September 2,

1861. He had enjoyed pretty good health in his youth. Six months previously, while in Russia, he fell ill of a fever unpreceded by premonitory symptoms, or by any change in the use of his faculties or the performance of his functions, or by febrile malaise, or the least general perturbation. His pulse became extremely frequent, and almost persistently so, from 120 to 130.

His appetite was better than it had been, and yet he did not gain flesh; he lost flesh, on the contrary, although his digestion was good, and he had no intestinal disorder. He would not have thought himself ill if it had not been for the constant frequency of his pulse (120). He tried quinine at Wilna, without any good results; and in Germany he was recommended the Kreuznach waters, in order to bring back to his skin herpetic eruptions, which he had had in former years, but which had long since disappeared.

He took the Kreuznach waters for six weeks. They produced some increased excitement, and while he was going on with his treatment, in August 1861, the prominence of his eye-balls was noticed, together with some injection of his conjunctivæ, especially the right one. On his return to Chartres, he consulted Dr. Roque, who diagnosed exophthalmic cachexia, and advised him to go to Paris.

Dr. Cazalis introduced him to me, and drew up the following account of our consultation:—

"M. X. is of medium size, very thin, dark, and without cachectic hue. On looking at him, one is at first struck with the prominence of his eye-balls, which, together with the marked dilatation of his pupils, gives to his face a strange undescribable expression. He related to us his previous history, as given above, and we then noted the following facts:—

"1. His pulse is from 120 to 125, equal, regular, but *very frequent, very small, and contracted.* The walls of the artery seem scarcely to yield to the heart's impulse, and resist expansion.

"2. The heart is ascertained to be of *normal* dimensions by percussing the præcordial region.

"3. The heart's impulse is very powerful; its rhythm is good, but it beats in a rapid hurried manner, almost convulsively. The powerful thump of the heart against the chest-walls contrasts with the smallness of the pulse.

"4. Both sounds of the heart are free from murmur, but we think we can occasionally detect some hesitation in the second sound.

"5. Although no friction-sound and no blowing murmur are heard over the heart itself there is friction-sound heard over the ascending aorta and the arch, and behind also, over the thoracic aorta. The same sound may be detected about the origin of the cervical arteries.

"6. Auscultation of the chest discloses unexpected signs. The patient has neither cough nor dyspnœa; he has never had asthma, and yet all over both lungs, although in a varying degree, the respiratory murmur is found to be loud, almost sibilant, expiration loud and prolonged: in a word, sibilant rhonchi are heard as in the most marked instances of humid asthma. Now, is this pulmonary condition to be ascribed to the present complaint? We believe not, and we are inclined to think that the state of the lungs is a consequence of iodism. We base our opinion on the fact that the patient's system has become saturated with iodine from the use of the Kreuznach waters, and that similar symptoms have been noted in persons that were not asthmatic, but were under the full influence of iodine.

"7. The eye-balls project considerably from the orbits, and the patient is himself aware of the change in his appearance. The right eye is more prominent than the left. The pupils are largely dilated. There is great alteration of sight; it is less distinct, the images are obscured, badly defined, and surrounded by a mist.

"The eyes are full of tears; the right conjunctiva especially is very much injected: there is true ophthalmia. We ascribed these phenomena to iodism.

"8. The thyroid gland is markedly hypertrophied, especially in the lower part and in the right lobe. The enlargement does not yet interfere with the neighbouring organs.

"9. M. X. has always had a good appetite, but has a still better one since his present illness set in. Digestion is excellent, but yet there is considerable and increasing emaciation.

"10. There is pretty marked thirst; the amount of urine passed is proportionate to that of the liquids taken; the secretion is of normal tint, and contains neither sugar nor albumen.

"11. The patient sleeps badly now, but used to sleep very well. He wakes three or four times in the night, which is a very unusual thing with him; and he is some time before he drops off to sleep again. Since he has been to Kreuznach, he has been worse in that respect.

"12. The patient says that he feels in a peculiar excited state which he cannot define, and his friends attest the same thing. Since he drank the Kreuznach waters, his speech has become jerking.

"13. The patient's sexual power had failed a little, but shows a tendency to recover itself."

I need scarcely call your attention to the completeness of this case. The three great symptoms of Graves's disease are there present; there is impaired nutrition, in spite of a perfect digestion; there is irritability, a jerking speech, an habitual febrile condition, and there are differences of resistance and

fulness between the carotid and the radial pulse. The case is a perfect type of this complaint, and presents a complete picture of Graves's disease in its developed state.

I mentioned that, in this case, the enlargement of the thyroid gland was not very marked. When this gland does not increase in size, or when the eye-balls do not become prominent, the disease may be said to be incomplete, as one of the chief symptoms is absent. This happened in two cases, which I will relate to you, one of which occurred in my private practice, and the other in a patient under my care at the Hôtel-Dieu. One of the chief symptoms is absent in each case; the disease is incomplete, but its existence is unquestionable, as you will be able yourselves to judge from the combination of the other symptoms.

Mrs. X. (from Jura), aged 38, and married for the last seven years only, consulted me on October 23, 1861. She enjoyed pretty good health up to the time of her marriage. In December 1857, she was attacked with a continued fever, with daily remissions, so much so that intermittent fever, recurring at the same hour every night and lasting till the next morning, was diagnosed, and that for a whole year. She complained at the same time of violent pains in the head, of distressing and persistent want of sleep, and of a difficulty of breathing which compelled her to spend a part of the night in an arm-chair, with an open window. She coughed, but did not expectorate anything. Subsequently, the fever still keeping on, she had violent palpitation of the heart, which scarcely ever ceased day or night. She never had a sensation of throbbing in the neck or head. Her eyes had begun to swell a few months after the fever set in, and before she had felt any palpitation. At the end of five months, the exophthalmos was at its maximum, and persisted to the same marked degree until the autumn of 1860. It is stated that the exophthalmos has been paroxysmal, that the eye-balls diminished in size for a little time, and then quickly increased to a very large size again. There was at such times pain in the eye-balls; the patient was unable to work on account of slight mists before the eyes, and the right eye was more prominent, but less affected, than the left. These paroxysms were very marked on several occasions, but the patient cannot say whether there was simultaneous increase of the other symptoms. The exophthalmos has gradually diminished for the last year, and the prominence of the eye-balls is not at present unpleasantly marked. The palpitation and the fever ceased before the prominence of the eye-balls became less.

The catamenia went on diminishing by degrees from the beginning of the complaint, and stopped for eight or ten months, but are now regular.

There was loss of appetite, diarrhœa, and extreme emaciation during the continuance of the latter.

Such was the patient's previous history. When I saw her, her complexion was fresh and her face full; this contrasted curiously with the extreme emaciation of the rest of the body, and with the complete atrophy of the breasts. The eye-balls were prominent, not painful, and there was slight double external strabismus.

No trace of goître; the throat was, on the contrary, markedly thin.

On careful examination of the heart, its size and the rhythm of its pulsations were found to be normal; no bruit was heard over it; the pulse at the wrist was normal, but somewhat frequent, 88.

Every summer the patient is seized with fever again, and has a persistent cough; but auscultation detects no organic lesion of the lungs.

There could be no doubt that this lady had been suffering from Graves's disease for some years past. Her feverish condition, her continued want of sleep, her irregular menstruation, disordered digestive and nutritive functions, her palpitation, unexplained by an organic lesion of the heart, and the prominence of her eye-balls, set all doubt at rest, although a chief symptom was absent, namely, hypertrophy of the thyroid gland. The disease, in such a case, may be said to be incomplete, but it exists nevertheless. The following case will give you another illustration that one of the principal symptoms, the exophthalmos, may be absent, and yet the diagnosis not be doubtful.

A woman, aged 29, was admitted into St. Bernard ward, on October 18, 1861. She was born at Dijon, and has lived there for a long time. For three years previously, she got out of breath whenever she went up a staircase or made an effort. She has been five years married, generally menstruates regularly, and is the mother of two children. She enjoyed good health during pregnancy, and states that her breathing improved very markedly while she was in that condition, but became still worse than before a few weeks after delivery. She had at such times palpitation of the heart, although she could continue to attend to her household work. A month previous to her admission into hospital, her breathing became very bad. and her palpitation increased; she noticed also that her throat swelled, and she slept badly. Her eye-balls did not become prominent, but her eyes had a strange restless look, and were constantly on the move.

She continued to nurse her child, who was now four months and a half old, although she had not much appetite, slept very

little, and was obliged to spend whole nights in the sitting posture, as she was seized with dyspnœa whenever she lay down. She did not lose flesh sensibly; and her child looked in good health.

Six weeks ago, she felt a violent pain and throbbing inside her head, which made her shriek; she became very impatient also.

On admission, she was in the following state:—

The thyroid gland was very markedly prominent, especially its right lobe; it pulsated and seemed as if it were thrust forward at each systole of the heart; a slight bellows-murmur could be heard over it, but not over the arteries in the neck. The patient felt her goître pulsate, and stated that her throat began to swell six weeks ago, that is, three months after delivery.

There was no exophthalmos, but the eyes were shining and very mobile. The same sensation of throbbing was complained of in the eyes as in the goître.

The heart's impulse was strong and frequent, and the patient had a pain in her back. The relative cardiac dulness measured 9 square centimetres ($3\frac{3}{5}$ inches); there was no cardiac bruit; the valvular click of both sounds was normal, perhaps a little accentuated and drier.

The pulse at the wrist was frequent, not full.

The patient coughed, and had occasionally some fever, and expectorated as in a mild attack of bronchitis. There were only a few scattered moist rhonchi, without any relative dulness at the apex; there has never been hæmoptysis.

The patient was extremely agitated and very irritable.

The baby was sent out to be nursed, but after that the patient's symptoms seemed to increase, and her eye-lids to get more widely separated.

The diarrhœa has stopped under treatment, but the appetite continued bad.

For the first case which I related to you, that of the gentleman under the care of Dr. Cazalis, there was scarcely any hypertrophy of the thyroid gland, although the complaint dated several months back. In the second case, goître did not show itself; although the disease had lasted several years. Lastly, in the third case, the patient had no marked prominence of the eye-balls, but merely a strange look about the eyes; in a few weeks from this time, perhaps, this symptom will show itself.

I have placed these three cases together, because they prove how irregularly goître and exophthalmos occur in Graves's disease, and because they show that one of the principal symptoms of the complaint may be absent, and yet all the others be present, so as to characterise it.

I must add, gentlemen, that I believe that exophthalmic goître may exist in a still more undeveloped condition, and that the disease may be foreseen, and does really exist, in a great number of instances, without there being exophthalmos, bronchocele, or extreme frequency of the pulse. I am glad to find that my excellent friend and colleague, Dr. Teissier, professor of clinical medicine in the Lyons School of Medicine, shares my views on this point completely, from his having seen incomplete cases of the disease. Dr. Teissier has noted four times absence of exophthalmos in patients who had palpitation of the heart, with swelling and enlargement of the thyroid gland, acceleration of the pulse, nervous restlessness, sleeplessness, and shining strange-looking eyes, in a word, in persons who exhibited most of the symptoms of Graves's disease. In these four patients, as well as in others, Dr. Teissier noted also a symptom well worthy of attention, namely, a rise in temperature of which the patient complained, and which could be measured by the thermometer. The increase has often been of one or two degrees centigrade, the normal temperature being 35° or 36° C. (96° to 98° F.). This symptom had not escaped Basedow, and you need not be surprised at its being present if you recall to mind that certain lesions of the sympathetic nerve are followed by a rise in temperature. You are also aware that in other neurotic affections, as in diabetes mellitus, for example, the patient often complains of a sensation of great heat, especially at night, and sleeps lightly covered only.

There is just now at No. 2, in St. Bernard ward, a woman suffering from Graves's disease, who presents all these symptoms: her pulse is usually 120, and her skin always warm and dry. During the periods of exacerbation, the pulse rises rapidly to 140 and 150 pulsations; the sensation of heat becomes unbearable, and the patient throws off all her coverings from her bed.

I have noted besides, in this woman, a symptom to which attention has not yet been called, and which I should like observers to look for, namely, the *cerebral macula*. If the epidermis be slightly irritated, after two seconds at most, a beautiful red stain is seen, which lasts nearly a minute. I can hardly believe that there is not in this case very marked *asthenia* of the *vaso-motor nerves*, in consequence of which the capillaries dilate rapidly, easily, and persistently, under the influence of the slightest irritation, just as happens in cerebral fever and in some ataxic cases of typhoid fever. Now, my opinion is that these three phenomena: acceleration of the pulse, rise in temperature, and cerebral macula, are of the same kind, and are traceable to the same cause, namely, some deep modification affecting the sympathetic and vaso-motor nerves in particular,

which gives rise to this artificial febrile condition and its usual concomitants, frequency of pulse and rise in temperature. As to the change in the sympathetic nerve, I shall presently discuss its nature with the aid of the experiments that have been made by Claude Bernard and by Schiff, but I will at once state now that I believe it to consist in asthenia, if not a momentary paralysis, of the vaso-motor nerves.

Graves's disease begins with an extraordinary nervous irritability, marked changes of temper, frequent flushing of the face, a sensation of fulness in the head, the eyes, and throat, and with violent throbbing of the heart. These symptoms come on in paroxysms which last from a few minutes to a few hours, and even days. Menstruation, in women, becomes disordered; the menses generally diminish in quantity, flow at long intervals, and even stop entirely.

The digestive system soon exhibits symptoms of disturbance; anorexia replaces bulimia; the patient complains of violent throbbings in the pit of the stomach, and has vomiting. In the majority of cases, the patient grows thin, even when the appetite is very good; in some rare instances, as in the case of a lady whom I saw in consultation, certain organs get developed, as the mammæ; while no indication is to be derived from the thyroid gland or from the eyes, and the emaciation of the rest of the body presents a strange contrast to the increase in size of certain parts. There must be, in such cases, partial hypertrophic congestion, probably due to a functional disturbance of the vaso-motor nerves. These premonitory symptoms may extend over several months and years, and sooner or later, the three prominent symptoms of Graves's disease show themselves, setting all previous doubts at rest as to the nature of the symptoms.

I will now describe the order in which the chief symptoms of this complaint develop themselves. The order is more apparent than real. I believe that all phenomena which are due to the same cause should appear simultaneously, and have the same period of invasion. This evidently happened in the case of a patient whose history I shall relate to you further on, who felt for the first time and on the same night, after violent mental emotion, strong palpitation, swelling of the thyroid gland, and exophthalmos, with copious epistaxis. It merely happens that the patient or the practitioner cannot always detect the phenomena. Palpitation first attracts attention, because the least disturbance of the heart's action cannot escape notice, and in addition to the throbbing at the heart, a marked sense of oppression comes on. The patient can no longer attend to his work, and is prevented from doing so by his palpitation; mental emotion also increases it. He complains of this, then,

before the strange look about his eyes, the protrusion of his eye-balls, and his capricious temper have yet attracted notice.

The exophthalmos is slowly developed, but when it has once set in, it continues and nearly always makes very remarkable progress.

Since, as I have told you, the hypertrophy of the thyroid gland is sometimes but slightly marked, it is very natural that it should be noticed at a late period. It has been forming for a long time already, when the patient first begins to speak of the increase in size of his throat, which he often remarks only when he has some difficulty in buttoning his collar. But if you carefully examine with the hand, and compare the size of the two sides of the throat, you will often be able from the very commencement of the disease to recognise in the right lateral lobe of the thyroid a marked difference which the patient has not yet observed.

Palpitation is to the patient the first symptom of the disease, and it is only at a later period that the exophthalmos and goitrous swelling show themselves.

There are two very distinct forms of the complaint, a rapid or acute form, and a slow or chronic one. They both have paroxysms, when the patient suddenly feels great oppression; the palpitation increases in violence, the eye-balls protrude more, the goître is more prominent, and the dyspnœa is so great as to threaten suffocation. These paroxysms are peculiarly grave in the acute form of the disease, and may put the patient in great peril of his life. The following case, of which Dr. Labarraque took careful notes, will show you to what extreme measures a practitioner may be obliged to have recourse, in order to save life in the acute paroxysms of Graves's disease.

"T., aged fourteen years and a half, of a good constitution, but of a somewhat lymphatic temperament. He went to school when he was twelve years old, and joined in games usual among boys of his age, and could swim without ever feeling the least difficulty of breathing. More than two years ago, however, it was noticed that he could not bear sea-bathing, except for a very short time: river-bathing had not the same effect on him.

"Some time afterwards, about eighteen months ago, he complained of some marked alterations of sight: he became more and more short-sighted, and this myopia, which has lasted up to this time, came on within a few weeks. He could not see what was going on at the board, in the class-room, and had to wear No. 9 glasses for short sight.

"In the beginning of August 1860, he went away for his holidays, feeling nothing particular at that time, and merely looking a little less well than usual.

"About a week after his arrival at Vierville, his friends noticed that his throat was rather full, especially at the base, but there seemed to be nothing serious about this.

"A few days afterwards, the fulness, having markedly increased, attracted greater notice.

"From that time, he could not bathe in the sea, as he used to be so fond of doing. The first time that he attempted it, as he plunged into the water, he felt violent dyspnœa as if he were going to choke, and he could scarcely manage to get away. He took a very hot foot-bath, had sinapisms applied, and the paroxysm ceased.

"But even at that time, although the swelling of the throat was getting gradually larger and larger, there was no marked difficulty of breathing.

"Dr. Lebâtard, of Trouville, who was consulted, prescribed syrup of iodide of iron internally, and iodide of lead ointment to be rubbed on the swelling.

"This treatment was carefully followed, but without any good results. The disease seemed to get worse, on the contrary; the swelling went on increasing, and respiration became impeded. The face altered, and assumed the dead white hue of commencing asphyxia.

"Towards the end of the holidays, the treatment was suspended, and after a few days the boy began to improve markedly; indeed, he was so much better that he was preparing to go back to school.

"At the end of eight days, the disease began to progress again, at a fearful rate: the anterior aspect of the throat continued to swell, almost visibly; the respiration became more obstructed and whistling, and the boy could neither run nor go up a staircase, and could scarcely walk at all.

"Dr. Blache, who was then consulted, expressed anxiety about the case. He stopped the treatment by iodine, enjoined rest, and ordered a weak ammoniacal ointment to be rubbed into the swelling, and frequent immersions into hot water of the hands and feet.

"Three days after this, there were such threatenings of suffocation that Dr. Blache was sent for in the middle of the night, and on his arrival desired to have a consultation with Dr. Trousseau. A few hours afterwards a consultation was held by us three."

The disease, as you see, gentlemen, had set in three months previously in this case, in August, and it was in the beginning of November that an acute paroxysm came on, without any appreciable determining cause, and threatened the boy's life. The face turned blue, the vessels of the neck swelled out, there was intense dyspnœa and imminent asphyxia. Tracheotomy

being indicated, Dr. Demarquay was asked to hold himself in readiness to perform it; but it was decided that, before having recourse to this extreme measure, the boy should be bled at once, and ice be applied over the front of the throat, and digitalis administered every hour. This plan answered all the indications, namely, of unloading the general venous system, of diminishing the fulness of the thyroid gland, and of quieting the extreme agitation of the heart.

The dyspnœa continued to be great during the day, but the symptoms of asphyxia disappeared, and the boy, who had not slept for the last eight days, slept on the following night for eight hours without waking. All present danger being removed, we waited for the good results of a continued sedative treatment, but we did not feel perfectly free from anxiety, and we asked the surgeon still to hold himself ready to act. When the boy woke the next morning, he was still agitated, as is so often the case when respiration is not free in consequence of some obstruction to the entrance of air. The agitation soon grew less, and three days after our first consultation, the boy could be said to have come to life again. He had no dyspnœa, and could, in our presence, go up or down stairs without experiencing any difficulty of breathing. The amelioration became persistent from the third day, and went on increasing; three weeks afterwards, T. could walk three miles to come and thank me for having attended him. By this time, his goître has almost entirely disappeared, as well as the exophthalmos, and he has no palpitation.

I will now, gentlemen, relate to you the history of a case of exophthalmic goître, running a chronic course, and of eleven years' duration.

Seven years ago, Dr. Labarraque was consulted at the Dispensary of the Philanthropic Society by a woman, thirty-nine years of age. She complained of difficulty of breathing, and palpitation of the heart; she had prominent eyes, and a very enlarged thyroid gland. She was treated successfully by bleeding, digitalis, and repeated doses of drastic purgatives. The exophthalmos and goître became less, and the palpitation ceased.

Dr. Labarraque had only a vague recollection of that case, as, when it came under his care, Graves's disease was not known as a morbid entity, when, a few days ago, he was consulted by the same woman about one of her children. She was not perfectly well, and still bore undoubted marks of her complaint. Dr. Labarraque kindly sent her to me, and on Sunday, November 18, I took down her history myself, of which the following are the chief points:—

She is forty-six years old. (Note, in passing, that exophthalmia

is rare at that age, and generally occurs from twenty to twenty-five.) In 1849, after a great fright (Stokes and Graves have mentioned fright as a cause of this complaint), she became subject from the day on which she was frightened to palpitation, which has continued ever since. Five or six months afterwards, her thyroid gland increased in size, her eyeballs became prominent, and she complained of short-sightedness. Her sight, although short, was at first excellent, and allowed her to work at lace-making, but amblyopia came on; she was troubled with muscæ volitantes, and saw large black spots on white grounds, so that she was compelled to give up her occupation as a lace-worker. At that period, that is to say, five or six months after, she became subject to palpitation; the catamenia stopped completely; her appetite was voracious, and she had diarrhœa at the same time.

The amenorrhœa lasted four months, and she then became pregnant. She was delivered on October 21, 1851, and recovered her health; she even got rid of her palpitation. She remained in this satisfactory condition until 1855, when she had an attack of pleurisy, after which the symptoms of her former complaint again showed themselves. Her appetite again became voracious, while diarrhœa returned, and she lost flesh and strength considerably. Dr. Labarraque, who saw her at that time, found that she had exophthalmos, an enlarged thyroid gland, and palpitation of the heart. She was treated by bleeding, drastic purgatives, and digitalis, and was enabled to resume her occupation at the end of eight or ten months.

In August 1856, her daughter got married, but she could not sign the marriage-contract, because of the great tremulousness of her hand, resulting from her extreme nervous excitability. At the present date, she still has prominent eyes, suffers from palpitation, and has an hypertrophied thyroid gland, of the *right lateral lobe* in particular.

Her pulse varies from 140 to 120 and 108; the carotids beat with considerable force, but the radial artery is merely frequent and of normal volume. This difference has been already pointed out by Graves and other observers.

I will now relate to you the history of a third patient, of a woman who was under my care in 1861, and whom I readmitted in 1862.

She is twenty-five years of age. She was born in the vicinity of Paris, and menstruated for the first time when thirteen years old. Her health has been pretty good up to the beginning of this year; but after the birth of her last child (her throat, I may observe, did not swell during pregnancy), she was violently moved on finding out that she had been right in suspecting that her husband was unfaithful to her. From that time, she had

violent palpitation of the heart; her eyes, according to the statement of her friends, assumed a strange aspect; they had been previously deep-sunken, but were now unusually prominent, and had a lustrous savage appearance. Soon afterwards, she noticed herself that her throat became gradually larger, whilst her appetite increased extraordinarily.

On her admission, I noted the strange look and the prominence of her eyes; she had a very developed goître; her heart beat with violence, and the impulse was visibly propagated into the carotids and into the enlarged thyroid. A continuous bellows-murmur was heard over the swelling which was raised en masse by the arteries at each contraction of the heart, while the hand applied over it had the same sensation of expansion as over an aneurism. The heart's impulse was violent; there was a soft bellows-murmur audible at the base, and prolonged into the aorta. The radial artery was of normal strength, and its pulsations were from 110 to 130. The patient had been taking iron, but it had to be stopped, because it increased the symptoms. Digitalis was given by itself, and ice applied to the swelling.

The eyes had the same savage look as before; the swelling of the thyroid was as marked, and the gland was still the seat of vascular throbbing and murmur, although the palpitation had diminished, and the cardiac bruit was less loud; when the patient was suddenly seized with vomiting, præcordial anxiety, and marked increase in all the symptoms of her complaint. The catamenia showed themselves on that day, but for a few hours only. I regretted afterwards that I was not informed in time of this last circumstance, because I would have tried by bleeding the patient at the elbow, or by applying leeches to her lower extremities, to increase the quantity and duration of the menstrual flow. Amenorrhœa had, perhaps, in this case, a large share in the production of the disease, for the patient had not menstruated since her last labour, although she did not nurse her child, and the first morbid phenomena showed themselves eight days after her confinement.

The paroxysm was perhaps a consequence only of the menorrhagic fever, that is, of the natural effort needed for the re-establishment of such an important function. Nearly complete aphonia set in after the paroxysm; but was it a mechanical complication, it may be asked; a result of increased congestion of the thyroid gland, or a purely nervous phenomenon, dependent on the general neuralgic condition of the patient, of which exophthalmic goître itself was only a consequence?

In 1863, I saw a young woman who had been formerly under my care for Graves's disease; she had suddenly lost her voice completely two days before, in consequence of some deep grief. This aphonia lasted six days, without any anatomical alteration

of the larynx being detected by means of the laryngoscope, and it disappeared suddenly, without any transition, after the pharynx had been slightly cauterised with nitrate of silver.

But to return to our patient: six days after the paroxysm which I mentioned just now, her eyes had a less savage look in them, the swelling of the thyroid was less voluminous, the bellows-murmurs were less loud, and the heart seemed to have become comparatively quiet. A short time afterwards, she was discharged in a satisfactory state compared with her former condition.

We have just seen the acute and the chronic form of exophthalmic goître. Each of them presents paroxysms which may return at long intervals only, of several months or several years, and may vary indefinitely as to their duration and gravity, or which may recur every month or several times a month. Perhaps when these paroxysms come to be better known, they will be found to have a certain relation to the hæmorrhagic molimen which takes place in the uterus every month; and if, on the one hand, it should be noted that in several cases amenorrhœa existed in the beginning, and, on the other hand, it be found that the symptoms abate, and the general disease improves, from the time that menstruation is re-established, or when the woman becomes pregnant, the practitioner will perhaps be able to deduce precious indications for treatment from these relations or those fortunate coincidences.

When the disease is fully developed, congestion of the eyes and of the thyroid gland may occur several times a day, concurrently with an increase of the cardiac pulsations.

The disease may go on increasing for several months, and then remain stationary for one or two years; paroxysms no longer show themselves, and the stage of decline then begins. The heart beats with diminished frequency and less intensity; the eyes become less prominent, and lose their savage expression, the bronchocele diminishes in size, is less elastic, contracts, and becomes harder; its erectile tissue, as Graves expresses it, grows less and less apt to be distended by the flow of blood. It rarely happens that the disease disappears completely; it merely recedes, and there always remains swelling and induration of the thyroid gland, with unusual prominence of the eye-balls.

The various blowing murmurs heard over the gland and the blood-vessels of the neck may disappear completely, as well as those of the heart. The improvement in the local is preceded by the disappearance of the general symptoms: the functions of the stomach and of the intestines become normal again, the temper ceases to be capricious, and the patient is able to resume his or her usual occupation. The disease, in females, is occasionally brought to an end by the re-establishment of the

menstrual flow or by the supervention of pregnancy. Some cases, then, do terminate favourably, but not all. The anæmia, which results from the disturbance of digestion, is sometimes so considerable that hectic fever supervenes, or the patient is so weakened that he becomes susceptible of any morbid influence, and dies of some intercurrent affection, which, in the majority of instances, has its seat in the respiratory organs.

Some, like Hirsch and Praël, have recorded cases which have terminated fatally in consequence of pulmonary, intestinal, or meningeal hæmorrhages. I have myself known an instance in which cerebral hæmorrhage proved the cause of death.

What is to be especially dreaded during the paroxysms is the danger which the patient runs of being choked. Under certain conditions, tracheotomy offers the only means of staying the progress of asphyxia, but the surgeon should not forget the extreme vascularity of exophthalmic goître, and should be on his guard against hæmorrhage which may prove *fatal* in a few minutes.

Before I speak of the pathological anatomy of exophthalmic goître, I will first relate to you the particulars of two cases which are of great importance, as showing the nature of this complaint. Stokes has perhaps laid too much stress on the existence of a cardiac neurosis, and he places in too subordinate a position the other phenomena of the disease compared with the functional lesion of the heart; hence the too great facility with which he is led to admit the existence of an organic lesion, namely, dilatation of the heart. Dilatation of the heart, when present, is accompanied by hypertrophy in exceptional cases only. I believe that in Graves's disease there may be a temporary hypertrophy of the heart, analogous to that met with during pregnancy. The first of the two following cases proves that although the complaint lasted two whole years, no persistent organic lesion occurred. The second establishes that, even when the disease is at its maximum, there need not be, in every case, dilatation, even though merely passive; and from this it may be inferred that the practitioner should not look upon a condition which is so often absent as one of primary importance.

The first of these two cases was communicated to me by one of my esteemed colleagues in the Faculty, whose daughter became affected, when eighteen years old, with exophthalmic goître and palpitation. She suffered from amenorrhœa also, and a disordered digestion, marked by violent hunger alternating with want of appetite and a dislike for food. This young lady's appetite is now regular; her exophthalmos and thyroid swelling have disappeared, and it is a fact worthy of notice that she recovered at a distance from Paris, in a

mountainous country where goître is endemic. She took very little iron, and her recovery is to be mainly ascribed to change of air, as it has been observed in other cases. She can take exercise with ease, and no longer has palpitation; in fact, she is perfectly cured, and her heart presents no appreciable organic change.

The second case was observed at Clermont (Oise), by Drs. Labitte and Pain. The patient was sent to me by Dr. Pain with a very detailed report of the chief symptoms which she had exhibited for two years, and of which the following is a summary:—

Miss X. has enjoyed very good health up to the age of fourteen. She showed great aptitude for learning, and, when a little girl, joined in her companions' games without ever feeling any difficulty of breathing. She menstruated for the first time at the age of twelve, without her health getting impaired in any way, and afterwards every twenty-eight days regularly. When she was fourteen years old, she seems to have menstruated with less regularity, about the months of December 1858 and January 1859, and to have had a very painful temporal neuralgia, which only yielded to cold affusions on the head. In April 1859, she had epistaxis, which continued for six weeks, and her catamenia stopped completely. From that time, Miss X.'s friends noticed that her eyes were getting larger and more prominent, and that her thyroid swelled in a remarkable manner. In September 1859, she went to Normandy, where she was born; her health improved a little, and there was a slight menstrual flow. Palpitation of the heart had come on simultaneously with the exophthalmos and the goître.

From October 1852 to June 1860, the three chief symptoms of the complaint continued to increase in severity, and according to the patient's mother, all the symptoms were markedly exaggerated towards the end of every month, from the 20th to the 30th. In June, she went again to Normandy; there was a temporary improvement, and the menses, which had been again suppressed since the month of October 1859, reappeared.

At the end of June, an alarming paroxysm occurred, with threatenings of suffocation, excessive throbbing of the thyroid gland, which had considerably increased in size, especially on the right side, extraordinary prominence of the eye-balls, violent palpitation, and blowing murmurs over the heart and the thyroid gland.

At the end of July, acute symptoms showed themselves, with fever and delirium.

During all this time, there was excessive hunger alternating with complete loss of appetite, and occasional vomiting.

The patient's voice changed from the time the exophthalmic goître first showed itself; sleep was disturbed by nightmares,

and the patient often started out of sleep, on account of a choking sensation. The eyes were incompletely closed during sleep, and there was a great flow of tears. The patient has never had ophthalmia. Her temper has grown very irritable; she perspired profusely, especially at the end of every month. The disturbance in the digestion, the want of rest at night, and her nervous excitement soon produced very great loss of flesh and extreme debility, which was still more increased by frequent diarrhœa.

Miss X. had frequent attacks of epistaxis, and her mother had noticed that the paroxysm came to an end after one of these attacks, which were sometimes attended with a copious loss of blood, and that all the symptoms improved. Steel was given at first, but without any good results, while digitalis was substituted with better effect. The determining cause of the disease was perhaps some deep emotion. No member of the patient's family was afflicted with goître, and there was no case of goître in that part of the country where she lived.

When I saw her myself, she was in the following condition:—

The exophthalmos was the most marked I had ever seen. The eye-ball protruded so much that, by asking her to move her eye about, I could see through the transparent conjunctiva the insertions of the internal, the external, and the superior recti muscles to the sclerotic. The eyes looked brilliant and savage; the ocular media were perfectly transparent; the pupil was still very contractile; there was no impairment of sight, and Miss X. could read at any distances large and small type, and her eyes could so remarkably adapt themselves to distances that she could be short or long-sighted at will. When she held the book which she read at the usual distance, or at a distance greater than the average one, the axis of her eyes did not alter, but when the book was held very near, double convergent strabismus was immediately produced, the pupils contracted, and she could read with ease.

The goître was of very large size, especially on the right side; very large veins were visible under the skin; the swelling was elastic, and arterial pulsations could be detected in it. There was very slight expansion of the swelling, but it was lifted en masse at each arterial diastole. Over it, continuous blowing murmurs were heard with rasping and sawing sounds.

There was violent throbbing, but no bulging, of the præcordial region; the heart's apex beat in the fourth intercostal space; the absolute dulness on percussion measured four square centimetres only.

No blowing murmur was heard; and there were only very dry and sonorous valvular clicks, like those which may be heard in young people who have just been running, and whose hearts'

sounds can be heard very distinctly through their thin chest-walls. The pulse ranged from 110 to 120; it had some strength, but was not in the least full.

Menstruation had been suppressed since the month of June, and there was profuse leucorrhœa. The appetite had become more regular for the last few days, and there was less diarrhœa. The emaciation was excessive; the skin, which was formerly transparent, was now brown and freckled in some parts; the complexion was pretty good. When Miss X. was attacked with epistaxis, her blood was of a pale rose colour, and stained linen yellow, showing her to be anæmic.

The treatment consisted in the administration of digitalis, in hydropathy, and the application of ice to the præcordial region. For several months, no improvement followed, but when I saw the young lady a year afterwards, she was stout and fresh-looking and in a considerably better state, although her eyes were as prominent and her goître as large as before.

Dr. Pain kindly supplied me with further details of this interesting case in June 1862.

Twice in the course of a year, there came on such a paroxysmal increase of the exophthalmos that one of the eye-balls became dislocated, as it were. The eyelids got behind the greater half of the circumference of the eye-ball, which had to be pushed back with a certain amount of force in order to get the lids to come forward again.

On several occasions (and this is a curious phenomenon, which makes congestion of the vessels of the thyroid gland and of the orbits resemble that of cavernous bodies), the goître disappeared suddenly and returned after a little while. This only occurred in the morning.

There was at times such a degree of nervous excitement that insanity was dreaded.

The patient's temper has now become gentle again, and there are no longer any congestive paroxysms; her complexion is good, and she is moderately stout; the pulsations of the heart have diminished, and I have been informed by Dr. Pain himself that the young lady has completely recovered for some months past.

I will now proceed with the general description of the disease.

I have told you already that in exophthalmic goître, the heart did not necessarily present organic lesions, but that some careful observers had noted hypertrophy of that organ, with or without valvular lesion (Praël, of Berlin, 1857), passive dilatation (Graves), either temporary, and noted only during the paroxysms, or permanent, when the disease had extended over a long period, and I have myself met with hypertrophy without valvular lesion.

Lastly, exophthalmic goître may affect individuals already suffering from heart-disease.

The pathological anatomy of this complaint is treated of in Dr. Withuisen's memoir. After expressing regret that Mackenzie, who has seen the only case of this disease which terminated fatally at the outset, does not give any details as to the anatomical condition of the organs which would have had an important bearing on the nature of the complaint, the Danish physician gives, in his memoir, a summary of seven autopsies. As this question is one of present interest, you will, I hope, excuse my entering into it at great length.

Withuisen remarks that the seven individuals whose bodies were thus examined had been ill for several years, and that the changes found may to a certain extent be regarded as consecutive. I quote his own words :—

"The best anatomical description is that given by Neumann. The patient, in that case, had died with symptoms of organic heart-disease and of a cerebral affection. The left ventricle of the heart was hypertrophied, without being dilated; the sigmoid valves were rigid, with thickened uneven edges; there was dilatation of the right heart without hypertrophy of its walls; the heart was fatty at the base. There were numerous atheromatous deposits in the aorta and the branches given off by the arch; the cerebral, ophthalmic, and ciliary arteries were likewise the seats of atheromatous deposits; the arteries at the base of the brain presented aneurismal dilatations here and there. The alterations in the ophthalmic and ciliary arteries had very probably a great deal to do with the loss of vision; which had occurred several months before death.

"The thyroid gland was of very voluminous size; it had a fibrous structure, and through it were scattered coagula of variable date; there were no cysts. The arteries supplying the gland were very much developed, especially the inferior thyroid, the coats of which were hard and fragile, and presented several aneurismal dilatations.

"The veins in the gland showed many traces of inflammation; they were partly obliterated and reduced to the condition of fibrous cords.

"The eye-balls were pushed out of their sockets by a considerable quantity of fat, but their size was also greater than usual, for the antero-posterior diameter of the left eye was 11½ French lines in length, and that of the right eye, 11 lines; the transverse diameters of both eyes were 11 lines long." Now, as Withuisen remarks, these diameters were greater than the normal ones, for, according to Mr. Sappey, the average length of the antero-posterior diameter is 10·6 lines, and that of the transverse, 10·1.

"There were numerous small extravasations of blood on the retina, and the choroid was of a uniformly red colour.

"Some time afterwards, Dr. Praël found in one case atrophy of the eye-balls, without increase of the cellular and adipose tissue in the socket. The thyroid gland was considerably hypertrophied; the left heart was dilated and hypertrophied; there were a good many atheromatous patches on the endocardium and along the arch of the aorta, and there was also aortic constriction and regurgitation. The patient's death was preceded by symptoms of softening of the brain, and this condition was found on examining the body afterwards.

"A patient died, while under the care of Dr. Henry Marsh, from gangrene of the lower extremities; and disease of the tricuspid and mitral orifices was found." It is not stated in Withuisen's memoir whether Dr. Marsh examined the eye-balls and the thyroid gland.

"Another case, observed by Dr. Smith (of Dublin), need be merely mentioned: the patient died of apoplexy; there were hypertrophy of the left heart and disease of the aorta. Basedow has recorded also the case of a man who, after having for ten years presented the signs of exophthalmic goître, died suddenly from cardiac disease. Dissection disclosed incompetency of the aortic valves. The thyroid gland was hypertrophied and full of cysts and of varicose veins. The eye-balls were atrophied, but were pushed forwards by a large quantity of cellular and adipose tissue. The same condition was found by Keusinger to explain the prominence of the eye-balls. He also found considerable hypertrophy of the thyroid gland in a man who had for years exhibited symptoms of Graves's disease, and died of a heart-affection.

"The seventh autopsy was made by Dr. Kœben, who found dilatation of the heart without valvular lesion in a similar case, and increase in size of the thyroid gland, a great many cells of which were filled by a gelatinous matter. The eye-balls, which had been very prominent during life, had sunken into the orbit after death, although the left orbit contained a sufficiently considerable amount of fat to prevent retrocession of the eye."

These reports of post mortem examinations, which Withuisen has brought together in his memoir, give you a great many of the elements of the question. Although some organic heart-disease was found in every one of these cases, I think that it would be exaggerating the importance of these lesions if too great a share in the production of exophthalmic goître were to be ascribed to them. In this disease, I repeat, I believe that the heart may, in the majority of instances, present variable and temporary alterations, analogous to those which are met with

during pregnancy, and it is in rarer cases that the cardiac lesion is permanent, only when the neurosis has been of prolonged duration.

The thyroid gland is very remarkably altered in structure.

It normally is so very vascular that Graves regarded the facility with which the gland becomes congested under the influence of the heart's action as a sufficient reason for comparing it to erectile tissue. The gland is, indeed, supplied by four large arteries, and occasionally by a fifth additional vessel which comes directly from the aorta. The veins of the gland are also very developed, and we have proofs of this excessive arterial and venous development whenever a surgeon carries his knife through the thickness of the gland.

Now, in cases of exophthalmic goître, the thyroid arteries increase in diameter and become flexuous; their extremities and branches enlarge considerably, and their anastomoses seem to multiply (Basedow, Stokes, and Hirsch). This exaggerated development of the arteries resembles the vascular dilatation known under the name of cirsoid aneurism, and accounts for the blowing murmurs heard over the swelling, and for the expansion detected by applying the hand over the thyroid gland. I mentioned when describing the swelling, that it was lifted up en masse during the diastole of the carotids, and that it expanded outwards in consequence of the dilatation of the divisions and branches of the thyroid arteries. The venous system of the swelling is also very developed (Marsh); large veins ramify on its surface and throughout its thickness (Henoch). When the disease is to terminate favourably, the swelling, as I have told you already, becomes less elastic and harder; in such cases, dissection has shown that the vascular system decreased, while there was an increase in the connective tissue which had become fibrous; small sanguineous cysts have also been seen which have undergone various metamorphoses. In the case recorded by Dr. Kœben, the thyroid cells were filled with some gelatinous matter.

All observers must have been struck with the exophthalmos, and hence attempts have been made to find out the anatomical cause which gave rise to this prominence of the eye-balls. With that view, the eye-ball, the vessels of the eye, and the intra-orbital cellular tissue have been particularly examined. Mr. Follin could scarcely find, during life, by means of the ophthalmoscope, an increase in the vascularity of the choroid; nothing in the structures in the eye could account for the exorbitis. Mr. Broca was not more successful. The only lesions noted in the eye by Withuisen and Neumann have been deposits of pigment round the optic papilla, but there was nothing which could explain the exophthalmos. Stokes thinks that it may be due to

dropsy of the eye-ball, but does not give a single proof in support of his view. Romberg found elongation and dilatation of the ophthalmic artery. Mr. Fano noted in one case considerable increase in the size of the veins of the orbit, so great, indeed, that, when the eye-ball was pressed back, voluminous veins were seen to raise the upper lid en masse. With regard to the cellular tissue of the orbit, Mr. Richet has seen in an individual suffering from anasarca the general œdema disappear, but the cellular and fatty tissue in the orbit continue to be œdematous. It is not stated whether there was not, in this instance, a local obstacle to the venous circulation. Basedow, Hastinger and Kœben have found an increase in the amount of cellular tissue within the orbit. In a case which I am going presently to relate to you, I found myself enormous hypertrophy of the cellular and adipose tissue inside the orbit, which pushed the eye out of its socket.

When we find, however, that the prominence of the eye-ball may, in a great many cases, show itself rapidly in a paroxysm and then disappear, we are led to ascribe that condition to a violent and active congestion. Thus might be explained the easy production as well as disappearance of the exophthalmos. But if the repeated congestions become hypertrophous, that is to say, if frequent congestions increase the nutrition of the cellular and adipose tissue within the orbit, these tissues gradually increase in quantity, and, by pushing the eye-ball forwards, give rise to permanent exophthalmos. When I described the symptoms of the disease, I mentioned that serious disorders of the stomach and intestines had been noted, and pathological anatomy has shown that in some cases death had been brought on by hæmorrhage into the stomach, the intestines, or the lungs. Lastly, the liver and spleen may be gorged with blood, increased in size, and I have myself found hypertrophic cirrhosis in two cases of exophthalmic goître. The kidneys themselves have been the seats of grave alterations, fatty and amyloid degeneration, and have presented all the characters of Bright's disease. I need not add that we must make allowance for complications, and that, when there is an organic heart-affection, in a case of exophthalmic goître, the cardiac lesion is the cause of most of the passive congestions of parenchymatous organs.

I now pass on to the *differential diagnosis.*

There is no other complaint, gentlemen, which can be mistaken for exophthalmic goître, for there is no other in which the three great symptoms pointed out by Graves exist. The insidious and sudden invasion of the malady, its distant or frequently recurring paroxysms, its variable duration, its course and nature, are as many distinctive characters, and if each of the principal phenomena of this morbid entity be examined one

by one, you will find that they again facilitate the diagnosis of this complaint.

The exophthalmos is double, equal on both sides, and unaccompanied by strabismus, characters which distinguish it from all exophthalmos of orbital or cranial origin; the eye-balls are extremely mobile, and the eyes are lustrous, while this is not the case when the prominence of the eye-balls is due to an organic alteration of the heart, when the eyes are often dull and merely prominent. The eyes of short-sighted individuals have a peculiar appearance, which can be hardly described, but the date and course of the myopia will not admit of a doubt long.

Can hydrophthalmia be mistaken for exophthalmos? The former affection (supposing it to occur on both sides, which is an exceptional occurrence) may be recognised by the dilatation of the pupils, distension of the sclerotic by the effused liquid; the cornea bulges markedly in front of the sclerotic; the prominence of the eyes is due to the dropsy of the chambers of the eye, whilst in Graves's disease the prominence of the eyes is owing to their protrusion.

I need not dwell much on the differences in the origin, shape, and mode of increase, of exophthalmic goître, of goître proper, and goître occurring during pregnancy. The first of these may develop itself, apart from all the conditions which give rise to endemic goître; it reaches its greatest point of development in the right lateral lobe of the thyroid gland; it increases very rapidly, enlarges during the paroxysms, and is entirely made up of vessels, whilst, in endemic goître, the throat-swelling consists in an hypertrophy of all the elements of the thyroid gland. Lastly, iodine, which often cures endemic goître, frequently causes an enlargement of exophthalmic goître. Goître occurring in pregnant women is apparently due to pregnancy, while that of exophthalmos is seemingly cured by pregnancy, or reduced in size by the re-establishment of menstruation. I have perhaps dwelt too much already on these differential characters, but before I leave off this subject, I must remind you of the palpitation, the intensity of which constantly increases the prominence of the eye-balls and the size of the thyroid gland.

Exophthalmic goître has been termed a cachectic affection. But we must first know what is meant by cachexia in general, for it is a term which has been often used, and its meaning has varied much at different periods. Nowadays, cachexia is understood to mean a deep alteration of the system consequent on morbid causes long inherent in the individual's constitution. This profound alteration is accompanied with important modifications in the proportion of the elements of the blood. There is diminution of the red globules, and increase of serum and fibrin; this modification constitutes anæmia or hydræmia.

There are numerous cachectic conditions, produced by any morbid cause powerful enough, by acting on the system for a prolonged period, to bring on general debility and anæmia, such, for instance, as the scrofulous, the cancerous, or the syphilitic diathesis, or abundant hæmorrhages from constitutional causes, &c.

If this is what is meant by cachexia, can we admit the existence of an exophthalmic cachexia? We cannot when the disease is temporary and curable, but we can when it resists all our attempts at curing it. For it is very evident that the blood crasis becomes deeply modified under the influence of great and prolonged disturbances of circulation. Oxygenation of the blood is imperfectly carried on in the systemic capillaries of exophthalmic individuals, whose pulse ranges from 120 to 160 beats in a minute. And the disturbance in hæmatosis necessarily gives rise to anæmia which shall be all the more marked in proportion to the duration of the complaint.

The dyscrasia depends, to a great degree also, on the impairment of the digestive functions. I need merely remind you of the strange bulimia which coexists with progressive emaciation. Thus, disorders of circulation and digestion give rise to anæmia, and prolonged anæmia is followed by cachexia. But this cachexia is only the last term of a morbid series which begins with multiple congestions, while these are themselves due to a peculiar modification of the sympathetic nerve, as I hope to be able to prove to you presently. To sum up, exophthalmic goître is, in my opinion, a neurosis of the sympathetic, if not a complaint attended with a material lesion of the ganglionic nervous system.

This neurosis gives rise to local congestions, the proximate cause of which is a modification of the vaso-motor apparatus. This view is supported by physiological and pathological facts showing that there are local congestions of nervous origin. Thus, in chlorosis, a condition in which the nervous system and the blood-crasis are so deeply modified, we note flushings of the face and congestions of the uterus followed by hæmorrhages, constituting what I have described under the name of *menorrhagic chlorosis.*

In hysteria, an essentially neurotic affection, we find delirium, coma, and protracted convulsions, followed by such a degree of congestion of the brain as to justify the practitioner in having recourse to bleeding. Can the profuse sweating and the copious flow of urine which occur in this complaint be understood except as the consequence of great congestion of the perspiratory glands and the kidneys? Lastly, Graves questions whether the sensation of choking which hysterical women have, and which has been compared to a ball rising

in the throat, or to claws constricting the root of the neck, is not caused by sudden congestion of the thyroid gland. He states that several practitioners, whose scientific knowledge he valued, had been frequently struck with the swelling of the thyroid gland during hysterical seizures.

The congestion of the thyroid gland, in hysteria as in exophthalmic goître, might be due to nervous paroxysms influencing the central organ of circulation, or some peripheral portion of the vascular system. The heart's action has been occasionally found to be accelerated and tumultuous during hysterical fits.

In one of the cases which I related to you at the beginning of this lecture, that of a young lady, from Clermont-sur-Oise, the swelling of the thyroid gland occasionally disappeared of a sudden to return a short time afterwards.

Other instances of local congestions dependent on the nervous system might be cited. Thus, an acute pain is frequently attended with redness and perspiration of the face; mental emotions cause blushing; modesty, anger, and love impart to the physiognomy a peculiar expression which is due to congestion of the face and eyes.

Now the existence of congestion in exophthalmic goître cannot for a moment be called in question. The swelling of the thyroid, which increases or diminishes according as the heart's action is accelerated or slackened, the prominence of the eyes, their shining appearance especially during the paroxysms, the heat and moisture of the skin, the mental disturbances, are all phenomena clearly indicating the existence of a congestion. By a process of reasoning, one is led to admit partial congestions in other diseases. In nervous asthma, the oppression at the chest and the dyspnœa are accompanied by pulmonary congestions, and the rhonchi heard in the lung-cells and bronchi, and the critical expectoration which terminates the paroxysm, are proofs of their existence. The least mental emotion, a bright artificial light, can cause this local congestion to disappear, which is dependent on the nervous element that gives rise to the asthma.

I will relate to you in illustration of this the following case which came under Dr. Gubler's observation: An in-patient at the Beaujon hospital, a young man of medium size, whose health was generally good, and who had no goître, no emphysema, no nervous asthma of the ordinary kind, was from time to time seized with a fit of difficulty of breathing, during which he sat up in bed, clutching at the bars in order to assist his breathing, and had the aspect of a man threatened with asphyxia. His face became of a violet hue; his bluish nails and lips showed that his blood was venous, and his eyes, which were considerably injected, and opened very wide, protruded as in exophthalmic cachexia. These paroxysms of dyspnœa lasted a few hours, and

the attack did not extend over more than a day or two. After that, he recovered his usual health, and his breathing was then so little impeded that he one day carried on his shoulders, from the bath-rooms on the ground-floor up to the third floor, a patient who could not walk, and did not pant for breath more than the most healthy individual would have done under the circumstances. Dr. Gubler could never make out, by the most careful physical examination, the existence of an organic lesion whatever, either of the heart or great blood-vessels, or of the organs of hæmatosis. During the paroxysms of dyspnœa, the respiratory murmur became feebler, and the chest resonance less clear; here and there a few rhonchi were heard, but those symptoms were very natural, if we admit that there was an internal congestion like that which could be seen externally.

This, gentlemen, is a good example of temporary congestion very probably due to a nervous cause. But in inflammations also, we find local congestions. Thus in whitlow, the congestion is confined to the phalanx or to the inflamed finger, and in general, unless there be general reaction, the arterial throbbings are restricted to the affected part: the individual's finger is feverish, if I may use the expression. Articulations attacked with rheumatism afford instances also of limited arterial and venous congestion. Those are inflammatory congestions, it is true; but you can every day observe physiological congestions, which are frequently directly brought on by nervous agency. Certain descriptions, lascivious sights cause the rapid, immediate, and temporary congestion of the sexual organs. In a physiological condition, this congestion constitutes erection; in a pathological state, it is priapism. Does not the structure of the organs which serve for the function of generation indicate that they are intended for congestion? The vascular tissue is then disposed in a special manner, and is called by anatomists erectile or cavernous tissue, or plexus susceptible of erection, as in the case of the ovarian plexuses, as shown by Professor Rouget's beautiful researches. It is a temporary congestion which causes catamenia in women, and the phenomenon of rut in the lower animals, and, when a woman has reached the critical age, how can we account for the successive hæmorrhages which occur just when the function is on the point of ceasing, unless we ascribe them to congestion of the vascular system? Now, all these hæmorrhagic congestions depend more or less on the nervous system, for fright will be sufficient to arrest the menstrual flow or to interrupt the local congestion necessary for accomplishing the act of generation.

There are, then, temporary physiological local congestions of nervous origin. But if you will review with me certain phenomena of natural history, you will find among them proofs of

analogous congestions. In plants which reproduce themselves by buds, the sap, at a particular time, flows abundantly towards those parts which are to give off the buds, and gives rise, therefore, to a local congestion. If you place two cuttings from the same vine, one in an atmosphere of 4° or 5° C. below zero, and the other in a conservatory heated to 20° C. above zero, you will see the latter produce buds, while the former will give no signs of vegetative activity. Is not a local congestion produced by such a process?

In the lowest classes of animals, as the fresh-water polyp, the hydra is produced by gemmation. This mode of reproduction is accompanied by a local congestion which manifests itself by the growth of fresh polyps, of fresh hydræ, which in their turn give origin, by the same process, to new beings, and on the same trunk you will find several living generations.

The same thing happens in animals of a higher order; nature has disposed everything for the continuance of the species, and the phenomena of puberty, especially in the breeding season, manifest themselves by local congestions of the membranous combs and the webbed feet of salamanders, in the caruncles of the turkey-cock, the goîtres and cutaneous combs of basilisks and of dragons, and even in that kind of bladder which is seen in a camel's mouth at the season of rut, and which has been shown by Savi to be due to the projection forward of the distended soft palate. These singular productions are, no doubt, proofs of the general expansion brought on by puberty, and which determines a state of true erection in the caruncles of the turkey-cock and the comb of the cock, &c.

These temporary congestive phenomena may be observed in the female also. During the twenty-four or twenty-six days that she is laying eggs, a hen's crest is red, and her collar of a deep blue, but her crest gets wrinkled when she begins to sit over her eggs. Need I add that during the period of rut, in most female animals, the congestion of the genital organs manifests itself by a flow of blood and by an increase in the secretion of the glands which are annexed to those organs?

Since we find that in animals rapid congestions of a variable duration return regularly under the influence of a physiological nervous cause, can we not suppose that a morbid condition, which is characterised by rapid congestions, also varying in their duration, and recurring in paroxysms, may be due proximately to a modification of nervous influence, and should be consequently classed with neuroses? Besides, may not the congestion of the thyroid gland and of the eye-balls be regarded as a kind of pathological erection of those organs, and are we not justified by Mr. Claude Bernard's beautiful experiments on the sympathetic nerve in comparing the morbid congestions of exophthalmic goître to those abnormal congestions which that

learned physiologist produces at will in different regions of the body by irritating or by cutting branches of the sympathetic?

Exophthalmic goître is, in my opinion, a congestive neurosis; and it is a morbid entity, because it presents special phenomena: palpitation and congestions of the thyroid gland and of the eye-balls. It is a pathological variety of the great class of neuroses, with a paroxysmal course, and should be regarded as entirely distinct from ophthalmias due to organic diseases of the heart, while it cannot be confounded with goître proper, of accidental or endemic origin.

I will now relate to you the particulars of a case which seems to me to throw the greatest possible light on the disease which we are now studying. The history of this patient shows, indeed, the undoubted influence of deep mental emotion on the generation of exophthalmic goître, and some of the anatomical lesions found explain the influence of the sympathetic nerve on the functional disorders which are peculiar to the disease, and on the secondary structural lesions.

A woman, aged sixty years, is admitted into St. Bernard ward on July 3, 1863, suffering from highly marked exophthalmos, and with the following history: In 1856, that is to say, seven years previously, she lost her father, whom she had attended at the cost of great fatigue. This loss caused her very deep grief. One night, after she had been crying for a long time, she suddenly felt her eyes swell and lift up her eye-lids, her thyroid gland increase notably in size and throb in an unusual manner: she had at the same time violent palpitation of the heart. Simultaneously with the development of this train of symptoms, she bled copiously from the nose throughout the night. Four days after this, she consulted Mr. Desmarres, who recognised exophthalmic cachexia.

A year afterwards, she went to Africa, where she soon caught intermittent fever. She was admitted for this into the Algiers hospital, and there, while under Dr. Bertherand's observation, her goître, which had been very marked, diminished rapidly. The two other symptoms, palpitation and prominence of the eye-balls, continued, however, to the same degree.

The fever lasted for nearly a year, and brought on a cachectic condition, from which she never recovered completely. In January 1863, she had an attack of angina pectoris, which lasted a few hours, with radiating pains in the right shoulder. It seems that a fortnight after her arrival at Algiers she had œdema of the lower limbs and ascites, which disappeared after four or five days. In 1863, this dropsical condition recurred on several occasions, but without continuing. When she came under my care, she showed no trace of œdema or ascites, and was in the following condition:—

Eye-balls considerably prominent; the free margin of the lower lid is more than four millimetres distant from the transparent cornea, instead of being in contact with it. The upper eye-lid does not cover a segment of the cornea, as it usually does, and is more than two millimetres away from it. In consequence of the protrusion of the eye-balls, the eye-lids no longer form regular curves, but intercept between them an hexagonal space with obtuse angles. The patient is readily dazzled by a bright light, which makes her feel as if she were drunk; she is long-sighted in spite of the prominence of her eye-balls. On the night when her complaint set in, she lost her sight for a time entirely, and for nearly a whole year she was hardly able to bear artificial light. She was at that time unable to read or sew, but can now do both by using spectacles for long sight.

At the commencement of her illness, her eyes were still bigger than they are now. She could very imperfectly close her eye-lids, and even now, during sleep, her eye-lids do not entirely cover the eye-ball.

Her heart beats with force, but much less so than at the onset of her complaint; it is found, on percussion, to measure 13 centimetres in a longitudinal direction (about $5\frac{1}{5}$ inches), and 12 centimetres ($4\frac{4}{5}$ inches) transversely. There is no blowing murmur at the base or apex, systolic or diastolic; nor is any heard in the vessels of the neck, although the arteries pulsate with force.

Pulse 96; habitual dyspnœa.

The liver comes down a little beyond the false ribs.

The thyroid gland is of small size: there is no goître.

Some time after the exophthalmos set in, the patient seems to have had a ravenous appetite for more than a year; she was obliged to take some food every two hours. She had a copious diarrhœa at the same time.

She menstruated for the first time at the age of 20; she had been chlorotic for five years previously, but menstruation, by degrees, caused the symptoms of chlorosis to disappear. The patient was menstruating at the time when the exophthalmos first came on; the catamenia were suppressed on that night, and have not since shown themselves.

Her father died of epileptiform seizures, which had occurred for several years. On admission, she complained of neuralgic pain in the ophthalmic nerve, in the occipital, and the first two cervical pair of nerves. Since the commencement of her illness, she has had trifling epistaxis every month, and about the same period.

She was ordered digitaline and Baumé's bitter drops.[1]

[1] [The chief constituent of Baumé's bitter drops is the alcoholic extract of Faba sancti Ignatii, which contains, as is well known, a large amount of strychnine.—Ed.]

When she left the hospital in the month of August, she suffered less from palpitation of the heart, but her eye-balls were as prominent. She was readmitted on December 3, in a weaker condition.

Six days afterwards, she was suddenly struck with apoplexy after a few trifling cramps in the legs; she fell out of bed without uttering any complaint, and was picked up in a state of asphyxia, with rigidity of the four limbs. A few hours afterwards, the left side got better, but the right side continued to be powerless, although not rigid. The patient did not recover consciousness, and died in the most complete state of coma, twenty-four hours after the attack.

On examining the body, a large hæmorrhagic centre was found in the left hemisphere of the brain, near the corpus striatum and optic thalamus.

The heart was of very large size, the walls of the left ventricle being the most hypertrophied. The free edge of the mitral valve was thickened, but there was no constriction of the aperture or incompetence of the valve. The aortic valves were a little roughened along their free borders, but were perfectly competent.

The aorta was encrusted with a calcareous deposit along its arch, and there were atheromatous patches in its descending portion.

The vessels at the base of the brain presented, however, no appreciable alterations to the unaided eye; the capillaries in the vicinity of the hæmorrhagic centre, and in that centre itself, were examined under the microscope by Mr. Peter, and were found free from calcareous and atheromatous changes.

The spleen was of voluminous size, measuring 12 centimetres in one direction, and 6 in the other. The capsule was not thickened; the splenic tissue was firm, and on section the glomeruli of Malpighi were found to be hypertrophied.

The liver was of nearly normal size, but of cirrhotic tint, and in an incipient lobular condition; its fibrous capsule was thickened, the trabeculæ considerably hypertrophied, and the tissue of the organ indurated. On microscopical examination, the hepatic cells were found to be normal, but to have diminished in quantity, and the interstitial connective tissue to be hypertrophied.

The kidneys had not increased in size; their capsule was not thickened; they had a granular aspect, were red on section, and showed traces of interstitial nephritis.

The thyroid body was of very small size, and its lobes hard, almost of the consistency of scirrhus; they had a lobulated quasi-cirrhotic aspect, owing to the retraction of their fibrous elements. On section, the glandular tissue was interrupted, and, as it were, squeezed by trabeculæ of an extremely thick

fibrous tissue, of a mother-of-pearl colour, and creaking when cut.

The thyroid arteries were small, not flexuous in the least, and showed no calcareous or atheromatous changes.

The eye-balls were thrust out of the orbit by the amount of cellular and adipose tissue which nearly filled the socket, was redder than usual, and contained a good deal of fat. The ophthalmic artery was not tortuous, nor was it abnormally large; the eye-balls, when removed from the cushion of fat on which they lay, were not of larger size than in health. They were not altered in structure.

The bones of the skull were extremely vascular, and were of more than double their natural size; in fact, they were all hypertrophied.

The cervical ganglions of the sympathetic were carefully dissected and examined on both sides by Dr. Peter and Dr. Lancereaux, clinical assistants at the Hôtel-Dieu. The superior and middle ganglions were of normal size and aspect. But the lower one, especially on the right side, was not only of larger size than usual, but was much redder also; numerous vessels were seen to ramify on its outer surface and throughout its interior, when examined with a power of 50 diameters. When examined under the microscope, a great many vessels were seen in its interior, with a thick admixture of connective tissue, and, in the midst of its fibres, nuclei and fusiform cells. There were a great many fat-globules; the ganglionic cells were very few in number, small, and with a mulberry aspect; some of them were reduced to a mere granular condition; the nerve-tubes were in small numbers.

All these details were well made out on examining a transverse section, with a power of 300 diameters. A very close network of fibres and of connective tissue was thus seen to enclose pretty narrow spaces containing small nerve tubules compressed, and, as it were, crushed by connective tissue. The examination thus made out two facts, to which I wish particularly to call your attention, namely, predominance of the connective tissue, and diminution of the nervous elements.

The cardiac plexus showed no apparent alterations, except perhaps some redness of the branches forming it. The ganglion of Wrisberg was unfortunately destroyed on an assistant cutting through the aorta. This case seems to me to be in the highest degree interesting, on account of its mode of invasion, its progress, the pathological changes found after death, and the probable connection of the lesions. It shows, in the first place, the immense influence of violent mental emotion on the production of exophthalmic goître. In the course of a single night, the three great symptoms of Graves's disease showed themselves:

palpitation, swelling and throbbing of the thyroid body, and exophthalmos. Of all morbid phenomena, congestion is the only one which can develop itself with such rapidity; and we have a proof that there were really multiple congestions present in the fact that the patient bled profusely from the nose at the same time, thus showing that there was simultaneously hæmorrhagic congestion of the pituitary membrane.

After a year, the swelling of the thyroid body disappeared, but the palpitation and exophthalmia continued, so that the disease became incomplete in its manifestations. This is far from being rarely the case, but it is interesting to see in the same person this complaint assume these various symptomatic forms.

I just now stated that the disease had set in with rapid and multiple congestions, but all congestion which does not disappear in a short time brings on hæmorrhage, effusion, inflammation, or what is termed hypertrophy. Now, this patient had frequent attacks of epistaxis and diarrhœa. Chronic congestions, in the majority of instances, cause interstitial plastic exudations, and from the organ having visibly increased in size, its parenchyma is supposed to have hypertrophied. The truth is, however, that there is, on the contrary, atrophy of the proper substance of the organ. For the plastic lymph becomes organised, is converted into fibrous tissue, and becomes a parasitical element, which develops itself by choking the proper tissue of the organ, or which is arrested in its evolution on account of its lower grade of vitality, and degenerates into fat. In other words, and in the language of the German school, hyperæmia may lead to exudation of a plasma in which the elements of cellular tissue get developed, namely, nuclei, fusiform cells and fibres: there is proliferation of the connective tissue, and then one of two things may happen, either the proliferation goes on, and the connective tissue becomes changed into fibrous tissue, which, from its exuberant growth, as much as from the retractile force with which it is endowed, determines constriction of the parenchyma, or it undergoes retrograde changes, becomes infiltrated with fat globules, and finally converted into adipose tissue. In the former case, cirrhosis is the result, in the second, fatty degeneration. Well, in the woman under my care, the cirrhotic process is the one which predominated, as shown by the condition of the thyroid body and of the liver. The tissue of the thyroid body was intersected by exceedingly thick fibrous partitions which compressed the globules, and gave rise to cirrhosis of the organ. The liver contained also a good deal of fibrous tissue, and the lobules were beginning to atrophy.

The kidneys exhibited what is termed interstitial inflammation, namely, an exudation of fibrin between the convoluted

tubules of the cortical substance, and perhaps Bright's disease would have come on, if the disease had lasted longer. The heart was evidently hypertrophied; its muscular fibres were more abundant than usual, and there was no predominance of fatty tissue in it.

We found, therefore, cirrhotic atrophy of the thyroid body, consequent upon great and prolonged congestion, incipient cirrhosis of the liver, hypertrophy of the heart, hypertrophy, with hyperæmia, of the cellular tissue within the orbit, and hypertrophy of the cranial bones.

I now proceed to enquire into the proximate cause of these congestions, and their consequences. We know that excision of the upper cervical ganglion is followed by hyperæmia of the ear, and that paralysis, or weakness, of the vaso-motor nerves causes relaxation of the coats of blood-vessels, blood-stasis, and consequently congestion. I can hardly believe that excessive grief did not cause in my patient a primary modification of her ganglionic nervous system. No appreciable change was found, it is true, in some of the cervical ganglions, but the lower ones, that on the right side particularly, were congested, and there was in the latter proliferation of the connective tissue, and diminution in the number and size of the nerve-tubules. Such a structural lesion must necessarily have interfered with the functions of the organ, and have been followed by consequences somewhat analogous to those caused by excision of the ganglions, that is to say, hyperæmia and all its results.

In conclusion, then, this autopsy authorises us to believe that the very numerous functional disorders which occur in Graves's disease are either due to temporary congestion of the sympathetic nerve or to a permanent structural alteration of the ganglionic nervous system; either of which becomes the origin of transient congestions or of irreparable lesions in various organs, which hypertrophy or atrophy in consequence of this by a process which I explained to you.

I regret not to have been able to examine all the divisions of the sympathetic nerve; my investigations were stopped by unavoidable circumstances, but I believe that a fertile mine remains to be explored in that direction, and I strongly recommend industrious men to make the attempt.

The *treatment of exophthalmic goître* has been complicated in the majority of instances. Stokes says that he has used with success lowering remedies and preparations of iodine. When the nature of a disease is not understood, its symptoms can be alone treated. Hence, against this form of goître, which was not recognised as a variety, iodine was used *internally* as well as *externally* by nearly all observers. But it was soon abandoned

by nearly everyone, as under this plan of treatment it was found that the symptoms grew worse.

Sir Joseph Oliffe has lately communicated to me the history of a young lady aged twenty-six, who for several years has been afflicted with exophthalmic goître. She was treated with iodine, which brought on a slight diminution in size of the thyroid body, but the eye-balls remained prominent, and the iodine caused very rapid emaciation and so great a debility that the patient was almost unable to take any exercise at all. She had only taken internally, however, about 25 or 30 grains of iodide of potassium in the course of three weeks. The medicine was dropped, and Sir Joseph prescribed antispasmodics and tonics, upon which the patient immediately felt better. It was only after the lapse of two months, however, that she regained her strength, and was enabled to resume her usual mode of life. The exophthalmos was as marked as ever.

Dr. Oliffe thought, and I might have done the same, that this young lady had suffered from iodism. I cannot hold that opinion now, however, although it has Rilliet's authority in its favour. For when we find that such a small dose of iodide of potassium as one-fifth of a grain a day is sufficient, at Paris and Geneva, to produce iodism, and, according to Rilliet, that a mere sojourn at the sea-side can bring on the same symptoms, I feel inclined to believe that the persons thus affected were the subjects of exophthalmic goître. Otherwise, how is the contradiction between everybody's experience and the cases of iodism published by Rilliet to be explained away, unless we admit the existence of a morbid element, which, under the influence of a certain remedy, gave rise to more marked manifestations? Iodide of potassium is given every day in large doses, in every country, at Paris as well as at Geneva, in doses of twenty to forty grains in the twenty-four hours. No accidents ever occur, although the drug be continued for several weeks in the same doses, and if, on the other hand, we should find that nearly infinitesimal doses bring on one of the chief symptoms of iodism—an increase in the size of the thyroid gland, with bulimia and various nervous symptoms—I am of opinion that such exceptional cases should be regarded as examples of exophthalmic goître.

Iodine, therefore, has, I believe, been wrongly accused by Rilliet. We all knew—and Rilliet knew it as well as we do—the great improvement which follows the use of iodine in ordinary goître; but it should be known also that iodine is a dangerous remedy to use in exophthalmic goître, and that it can give rise to paroxysms. When, in a case of goître, you find palpitation, protrusion of the eye-balls, and a strange look of the eyes, never give iodine. You have to deal with exophthalmic goître, and iodine will only increase all the symptoms of the disease.

In some rare cases, however, preparations of iodine are borne without any ill effects, and even with a semblance of improvement by persons suffering from Graves's disease.

About the middle of June 1862, Dr. Bruneau (of Villaines) sent to me a lady, who generally resides in Paris. Her history is of sufficient interest to deserve being related in some detail. She presents us, besides, with an instance of acute exophthalmic goître. She is thirty-five years of age. About the beginning of the year 1861, she felt curious sensations about her heart, which she compared to a kind of itching. Her heart, from that time, and ever since, has beaten with greater rapidity, and even after she had rested in my consulting-room for more than an hour, I counted 120 pulsations in the minute.

Since the month of February 1862, the catamenia have diminished in quantity, and become somewhat less pale. About the middle of March, however, she noticed that her throat swelled, especially on the right side, and she had pain in the eye-balls. Eight days later she noticed herself, like everybody else, the prominence of her eyes. She suffered from nervous irritability and difficulty of breathing, and although her appetite was markedly increased, she lost flesh. The bronchocele and the exophthalmos progressed so rapidly that, in the space of six weeks, they reached the point at which I saw them. The doctor who attended this lady in Paris advised her to go on a visit to her friends at Villaines (Mayenne), and to take daily twenty grains of iodide of potassium and some iron-pills. The influence of country air and perhaps the treatment made the patient regain her strength. The point, however, on which I wish to insist is that the goître diminished a little, in spite of the use of iodine in large doses, although, according to the patient's statement, the exophthalmos showed a tendency to increase. After this treatment had been carried out for a month, the disease remaining stationary, all remedies were ceased, and in a few days, the thyroid body increased again to its former size.

When I examined this lady, I found a large bronchocele (larger on the right than on the left side), and considerable protrusion of the eye-balls, the left of which was rather painful when pressed; she felt a sensation as if dust had got into her eyes. Strangely enough, she had become long-sighted since her complaint had set in three months ago, and she could not read or sew, except by holding the objects at some distance from her.

When I held the bronchocele between my fingers, I had a sensation of expansion, and on listening with the stethoscope, I heard over the swelling a double bellows-sound, which was single above it, on a level with the bifurcation of the carotid artery, and corresponded to the ventricular systole. This afforded a

proof that the double bellows-sound heard over the bronchocele was not merely due to transmission of sounds originating in the common carotid, since the arterial bruit was single.

There was no hypertrophy of the heart, nor any abnormal valvular bruit.

My object in relating this case was to show you that, although, in the great majority of cases, iodine exerts a bad influence on the exophthalmic neurosis, it seems occasionally to produce temporary improvement of the patient's condition. I would not, indeed, leave on your mind the impression that iodine is invariably hurtful in Graves's disease. I once attended with my excellent friend, Dr. L. Gros, who was the first, in France, to call attention to Graves's disease, a man, about fifty years of age, whose condition was singularly improved by the prolonged administration of iodide of potassium.

This case had not, however, converted me with regard to iodine, when I happened to see a case in which an error which I committed enlightened me.

In the course of October 1863, I was consulted by a young married lady, who habitually resides in Paris. She was suffering from a subacute exophthalmic goître. The bronchocele was of great size. When I examined her for the first time, although I had let her rest for a long while, and although I repeated the examination several times, and at sufficiently distant intervals, so as to make sure that she was no longer under the influence of emotion, I still found that her heart beat at the rate of 140 to 150 times in the minute. I recommended hydropathy, and I wished to administer at the same time tincture of digitalis, but, preoccupied with the idea that there would be some danger in giving iodine, I wrote iodine instead of digitalis, so that the patient took from 15 to 20 drops of tincture of iodine a day, for a fortnight. When she then came back to me, her pulse was only 90. I found out my mistake, and I substituted tincture of digitalis for that of iodine, but, after another fortnight, the pulse had again gone up to 150, so that I at once returned to the iodine.

Notwithstanding these exceptional cases, however, bear in mind that iodine generally does harm in Graves's disease.

Now, what does chemical experience say respecting the preparations of iron? The patient sometimes comes to you in a state of very marked anæmia, with a pale complexion, with œdema, bellows-murmurs over the base of the heart, extending up the vessels of the neck. Steel seems to be indicated then, and nearly all observers have recommended it. But read the cases that have been published, and you will find how little good steel has done, when it did not do much harm, and note that it was nearly always given together with digitalis, while the

patient was kept on low diet, and topical applications were used to the swelling to prevent congestion. The administration of iron would probably have been followed by still worse results, if it had not been counter-balanced by the influence of the other drugs, by digitalis in particular. As to me, I believe that iron does harm in exophthalmic goître, and you will concur with me if you recall to mind how we were compelled to stop it in the case of the woman who lay in bed No. 34, in St. Bernard ward, who soon became quieter, and had less palpitation when I substituted tincture of digitalis for the iron.

Dr. Graefe has before me pointed out the dangers and counter-indications of a treatment by preparations of iron. It should be avoided, he said, when there is considerable vascular excitement, and the pulse is more than 100 or 110 in the minute, because all the symptoms are intensified by it. We have seen that in some cases, however, the administration of iron is not followed by bad results, and the case which I related just now is an instance in point. Bear in mind also the happy results which I obtained in the case of the boy T. from bleeding, drastic purgatives, digitalis in large doses, and the application of ice to the thyroid swelling.

I can, from experience, recommend you to have recourse, in this singular affection, to bleeding, digitalis, and hydropathy. When I advise bleeding, I do not do so in an absolute manner, and, of course, not with the view of combating the anæmia and the nervous element of the disease. It is only with one end in view, namely, that of averting the imminent danger which may result from congestion of the thyroid body, of preventing asphyxia by depleting the blood-vessels, and of quieting palpitation. The first indication during a paroxysm is to prevent suffocation. This is effected by diminishing the size of the swelling: the continuous application of cold prevents the flow of blood to it, and artificial congestion may be induced in other regions, as the lower extremities for instance, by means of Junod's boots, large mustard poultices, &c. By and by, when the paroxysm is over, and there is no danger of suffocation, act on the supposed cause, on the nature of the disease. Exophthalmic goître is, in my opinion, a neurosis which principally affects the heart and the supra-diaphragmatic arteries; while Stokes thinks that it is pre-eminently a cardiac neurosis characterised by violent palpitation. Administer digitalis, therefore, the sedative par excellence of circulation. Be not afraid of giving it in large doses; yet, feel your way with it, and only stop when you have induced symptoms of incipient poisoning, when the patient complains of vertigo, of cephalalgia, and of nausea.

The pulse will also furnish you with indications when the medicine should be given in small doses, or should be dropped

altogether. When the pulse falls to 60 or 70, stop the digitalis, or diminish the doses.

When the patient's life was in danger, I have obtained good results from the administration of tincture of digitalis, given every hour in doses of eight or ten minims. You have not to fear in such cases the accumulating effect of the medicine. The boy T. took without harm 109 drops of tincture of digitalis in the space of only ten hours.

I will now tell you what good results may be expected of a treatment by hydropathy. Three years ago I was summoned to Crest, in the department of the Drôme, to see a lady who, for the sixth time during the last six years, presented all the symptoms of exophthalmic goître: prominence of the eye-balls, swelling of the thyroid, palpitation of the heart, pulsation, with bellows-murmur, of the carotids, obstinate vomiting, and congestion of the liver. In 1858, I was consulted again, and I advised hydropathic treatment. Dr. Gilbert-d'Hercourt superintended the treatment himself in his establishment at Longchêne. Bearing in mind, says Dr. Gilbert-d'Hercourt, in his relation of the case, that all the relapses of Mrs. B. had been preceded by a diminution or a complete suppression of the catamenia, he decided on carrying out the hydropathic treatment in such a way as to bring on congestion of the uterus, and thus produce a healthy revulsion. The hepatic engorgement soon disappeared; the protrusion of the eye-balls and the swelling of the thyroid became less and less marked. Mrs. B. was able to resume her ordinary occupations, and to sing for several hours without fatigue. In 1859, in the month of June, she had another relapse, or rather a fresh paroxysm, preceded by suppression of the menses. A hydropathic treatment again mastered the disease, and I have since been able to verify the perfect health of Mrs. B. She walks and sings without getting out of breath, she no longer suffers from palpitation; her pulse is not so frequent as it used to be; her appetite is good, digestion easy, and she sleeps well.

Hydropathy has several times been followed with the same good results in similar cases; it is a plan of treatment which should not, therefore, be neglected. You know what good effects may be obtained from it in anæmia, chlorosis, and hysteria, and you are aware also that many visceral engorgements have been cured by it. You must, therefore, think it quite natural that exophthalmic goître, which I have regarded as a congestive neurosis, should be favourably modified by it.

The continuous application of ice over the præcordial region, and to the thyroid body, is a powerful measure which I cannot too strongly recommend to you.

Now is perhaps the best time for dwelling on the therapeutic

indications, and for analysing the reasons which make certain measures successful in the treatment of this complaint. I will be brief, and will merely remind you that bleeding and revulsive applications to the extremities are employed against the congestion of the thyroid gland, avert the cause of asphyxia, while digitalis quiets palpitation, diminishes the frequency of the cardiac and arterial pulsations, and the hydropathic treatment offers the twofold advantage of causing a violent revulsion to the skin and of rendering innervation and nutrition more perfect. I should perhaps dwell more on the necessity of re-establishing menstruation. This is certainly an important therapeutic indication, but in order that it should succeed, one must wait till an hæmorrhagic tendency shows itself in the uterus. It would be bad medicine to try anyhow and at all times to bring back menstruation. One should know how to wait and to act only when nature seems to indicate it. Revulsives may then be had recourse to, and a few leeches be applied to the lower limbs, &c.

Lastly, if you have not succeeded in averting the paroxysm, and if it should be accompanied by a sense of choking threatening life; if revulsives and the application of ice to the swelling do not remove the risk of asphyxia, you may have recourse to tracheotomy. But bear in mind that the operation cannot be performed under more serious circumstances, and that the patient may die under the surgeon's knife. I have laid much stress already on the extreme vascularity of the thyroid gland in exophthalmic goître, and I have known a case in which death from hæmorrhage occurred during the operation: you should take every precaution, then, against hæmorrhage. With this object in view, Mr. Demarquay proposes the use of the écraseur, which has done so much good service since its invention by Chassaignac. One of the great advantages of this method is to secure one in nearly every instance against the grave hæmorrhages which so frequently follow upon the use of the knife in regions where ligatures cannot be easily applied.

The following is the manner of operating recommended by Mr. Demarquay: The thyroid gland is to be exposed by dissection, and all the subcutaneous and the sub-aponeurotic vessels liable to bleed should be secured by ligature; and after this the chain of the écraseur is to be passed underneath the isthmus of the thyroid. If the removal of the gland be accomplished in this manner without hæmorrhage, the trachea can next be divided, and a canula introduced into it.

Mr. Chassaignac thinks that the knife need not be used at all in this case, and he recommends instead that the skin be pinched in a transverse direction, and that all the soft parts situated in front of the trachea be included in the chain of the écraseur.

There would then be two stages in the operation: in the first, all the soft parts would be cut through by the écraseur; in the second, the trachea would be opened with a knife, and a canula introduced.

These are, as you may see, two different processes of one and the same method, namely, removal by means of the écraseur. This method offers the immense advantage of considerably lessening all risks of hæmorrhage, and future experience will pronounce on its value. But whichever mode of operating you may prefer, never forget to get ready beforehand all the means which medicine and surgery place at your disposal for arresting bleeding, which in these cases may in a few minutes endanger the patient's life.

LECTURE XX.

ANGINA PECTORIS.

Angina Pectoris symptomatic of an organic affection of the heart or of the great vessels.—In such cases, the organic lesions merely favour the development of the Neurosis.—Idiopathic Angina Pectoris, due to a rheumatic or gouty diathesis.—It may be a manifestation of Epilepsy, and may then constitute either a variety of Epileptiform Neuralgia, as is most frequently the case, or a variety of Aura Epileptica.—Angina Pectoris dependent on Graves's Disease.—Its invasion is sudden, its symptoms variable.—It may cause sudden death.—Treatment.

Gentlemen,—In spite of the numerous publications which treat of angina pectoris, the history of that complaint is not very satisfactorily known; and the various opinions which have been expressed as to its nature have thrown so little light on the subject that I wish, in my turn, to communicate to you my views concerning this singular neuralgia.

A woman, who died some time ago in St. Bernard ward, of aneurism of the aorta, furnished us with a remarkable instance of this complaint. Her attacks, which at first occurred at pretty distant intervals, recurred very frequently towards the last, and few among you have not had an opportunity of witnessing one of those awful paroxysms.

She was suddenly seized with an excruciating pain, without any appreciable determining cause, either while sitting motionless on her bed (the only posture which she could retain) or while moving. This pain started from the præcordial region, and radiated from it to the base of the chest, producing there a sensation of constriction which the patient compared to that which might be caused by an iron girdle tightened with force. It then spread to the loins, and, ascending towards the cervical region, attacked the left arm, and extended into the very tips of the fingers. The skin of the hand and fore-arm could be then seen to become excessively pale, and almost immediately afterwards to turn of a markedly bluish or livid tint. After the pain had ceased, the arm and hand felt numb for a few minutes. The pain was such as to make the patient cry out; her features were contracted, she sat in an upright position, as if dreading to be choked, although she breathed pretty freely. The paroxysm lasted a few seconds, and returned at intervals,

which grew proportionately shorter as the disease drew to a fatal termination.

In this instance, the angina pectoris was symptomatic of an organic lesion, and such was also the case in a patient about whom I was lately consulted by Dr. Périer.

He was a military superintendent, fifty-five years old. His attacks, which dated seven years back according to his statement, were chiefly characterised by a sensation of numbness and tingling in the skin of the left axilla, and spreading from there to the whole corresponding side of the chest. He often felt shooting pain, like that of neuralgia, but which was quieted by his squeezing his back against a resisting surface, as a piece of furniture, for example.

For the last six or eight months, he had become subject to some oppression at the chest. A somewhat rapid walk, the least active exercise, brought this back, and he was troubled with pain, even if he had been engaged in merely signing away many papers, in the discharge of the duties of his post.

On examining his chest, all the physical signs of aneurism of the aorta were made out. The action of the heart was violent without abnormal bruit; higher up and in front, a distant double bellows-sound was heard, and was audible also in the back all over the left side of the chest, but in the greatest intensity along the vertebral column, on a level with the spine of the scapula. Deep percussion also over the plessimeter made out dulness over the same spot. Vesicular breathing was perfectly normal all over the chest.

These two cases would seem to confirm an opinion held by some physicians, namely, that *angina pectoris* depends on the presence of appreciable organic lesions of the heart, of the great vessels, or of neighbouring organs. You are aware that Heberden (who was the first to give to this complaint the name by which it is now known, and who has left us a pretty good description of it), and, after him, Parry, Kreysing, Burns, J. Frank, &c. ascribed angina pectoris to ossification of the coronary arteries. Others, on the contrary, have referred it to hypertrophy with dilatation of the heart, ossification of the auriculo-ventricular or aortic valves, pericarditis, accumulation of fat on this membrane in the mediastinum or on the heart itself, displacement of this organ, compression of it by a tumour or through abnormal development of some one of the abdominal viscera, aneurismal dilatation of the aorta, inflammation of this vessel, mediastinal abscess, ossification of the costal cartilages, &c.

I do not deny that angina pectoris may coexist with one or other of these various lesions, and that it often (most often,

perhaps) is symptomatic, as has been said, of organic diseases of the heart or of the great vessels. But while, on the one hand, the variety of these lesions makes one suspect their etiological value, on the other, the numerous cases in which such lesions exist without the patient suffering from anything like paroxysms of angina pectoris, and, per contra, authentic instances of individuals who during life presented all the characteristic symptoms of angina pectoris, while, after death, dissection disclosed no anatomical lesion by which these symptoms could be accounted for, prove that this complaint is not essentially due to the presence of organic diseases.

From the absence of appreciable structural changes, and from the extreme variability of the phenomena, which I shall endeavour to describe to you, we must conclude that angina pectoris is a neurosis, or, to use a more precise term, a neuralgia. As to its seat, which some have placed in the diaphragm, others in the respiratory muscles, and most in the heart, this neuralgia generally affects the cardiac nerves given off by the pneumogastric, and radiates to the nerves of the cervical and brachial plexuses.

One of my oldest and most intimate patients, a lady, forty-seven years old, suffered in her youth from very obstinate chlorosis, accompanied by very acute neuralgic pain, which varied very much in its seat. For some years past, she has had very mobile rheumatoid pains, attacking sometimes the limbs, and at other times the viscera, and curious nervous disorders, which might be called hypochondriasis, if this lady were not a person of very great sense. I may add that her health is excellent, as far as the functions of organic life are concerned. For the last two years, she has noticed that, when she goes up a staircase pretty quickly, she is suddenly seized with an acute pain behind the sternum, rapidly extending to the left shoulder and arm, and causing trifling numbness. On her stopping, the sensations disappear in less than a minute. I have examined her heart and her lungs with the greatest care, I might say, with the most devoted solicitude, on several occasions, immediately after she had just had one of these seizures, and never at any time have I discovered in the heart's rhythm, in the valvular sounds in the region of the aorta, or in the lungs, the least sign, the least phenomenon, different from what is found in health, with the exception of some marked acceleration of the heart's action.

Quite recently, when I intended to speak to you of angina pectoris, I was consulted by a gentleman, aged forty-five, who had all the appearances of the most flourishing health. He took more than ten minutes to come up to my door, and, when in the ante-room, he dropped on a bench, looking pale, and in

a condition which frightened my servant. A few minutes sufficed to make him right again.

When, half an hour afterwards, he came into my consulting room, I could never have suspected from his blooming appearance what had so lately occurred. He then told me that fifteen years ago he had had a very bad attack of syphilis, of which he had not been well cured. Three years afterwards, he had a very violent and obstinate attack of sciatica, and, subsequently, pains in the limbs, of which he was cured, after many unsuccessful treatments, by iodide of potassium. Later again, he had had an attack of gout in the big toe. He had never passed any gravel, and there was no history of gout in his family antecedents.

The angina pectoris had begun a year previously. The attack was very slight, and only recurred when he took any violent exercise, at rare intervals ; in a short time, less active causes sufficed to bring on a paroxysm, which recurred at more frequent intervals. For some months, for the last month particularly, his life had become unbearable. If he happened to walk up the least ascent, he was instantly seized with pain, and was compelled to stop. He had just come from Lyons on the day when he consulted me. He had travelled all night, and as he came out of the railway carriage, he had to walk a few steps about the station to get a cab. Although he walked quietly, he was seized so violently that he had a kind of fainting fit, and was obliged to sit down in the mud. His travelling companions put him upon his legs again. The pain which he felt was excruciating ; it began behind the sternum, nearly on a level with the fourth and fifth ribs, somewhat about the region of the heart, which beat violently during the attack. It extended from there to the root of the neck, and to both arms equally, causing a painful sensation of numbness as far as the tips of his fingers. He fancied that his hands swelled a little at such times. He was then obliged to stop short, and to keep his chest motionless, dreading to draw in his breath lest he should increase the fearful constriction which crushed his chest. When the pain was more intense, he was seized with vertigo, and fell into a state almost like syncope.

The emotion which my examination caused him, and the movements which he made to take off, and afterwards put on, his clothes, sufficed to bring on a slight paroxysm.

It would certainly be difficult to meet with a more marked case ; and I confess that I felt sure I would find some grave lesion of the heart, or of the great vessels. But on the most searching examination, I detected no abnormal condition of the intra-thoracic organs. And as I have alrady met with a good many cases of this kind in the course of my career, and as

I have seen persons as gravely affected as this gentleman was get perfectly well, I must of necessity admit that angina pectoris, even when most intense, need not be a symptom of an organic lesion. Presently, when I come to speak of treatment, I will relate to you two cases of cure, one in a patient of Dr. Duchenne (of Boulogne), and the other in a patient of Dr. Aran. These will show you still more conclusively that angina pectoris may be only an idiopathic neuralgia, in the sense usually meant by this term.

A case, however, recently came under my notice and that of my esteemed friend, Dr. Marx, which shows that one should be very careful before affirming that no organic lesions exist. An ex-bill-broker on the Paris Bourse, who had been formerly subject to very severe hepatic colic, which had left him for several years, began to complain of choking sensations which came on suddenly whenever he took a little more active exercise than usual. The sensation of choking was accompanied by an acute pain behind the sternum, radiating to the left shoulder and arm. There was no habitual dyspnœa, and nothing could excite the suspicion that the *angina pectoris* was a symptom of an organic lesion. But auscultation afterwards detected the presence of an aneurism of the arch of the aorta which increased rapidly, and from that time, there came on habitual orthopnœa, and paroxysms of angina pectoris recurred on the patient making the slightest movement. Dr. Marx had one day spent a few moments with him, encouraging and consoling him, and had been accompanied by him on his going away as far as the bedroom door; but the doctor had no sooner got to the bottom of the stairs than he was hastily summoned by the patient's servant. On going up again in all haste, he found a corpse. The aneurism had suddenly burst into the trachea, and had caused fatal hæmoptysis.

In the month of September 1865, I was consulted by a patient sent to me by Dr. Lefebvre, of Roubaix, and suffering from angina pectoris. The complaint had set in suddenly about the middle of the preceding year, during an after-dinner walk, and the paroxysms had recurred several days in succession. They disappeared for some time, and then returned with greater intensity than ever, *at the same hour* invariably. They soon ceased from being periodic, and recurred under the influence of the slightest effort, or during sleep, on the patient starting up. At last, symptoms of a serious hypertrophy of the heart, with lesions of the ventricles, showed themselves. I will, therefore, willingly admit that, in some cases, even though the most careful examination will not be able to detect anything in the aorta or in the mediastinum, there are lesions present which become manifest at a later period. The same thing occurs in the case

of angina pectoris as in that of some obstinate intercostal neuralgias, the organic cause of which has been long overlooked, although it does not follow from this that even the most obstinate intercostal neuralgias are always symptomatic.

When I first began practice, I attended for several years a gentleman whose complaint I did not for a long time recognise, and whose case taught me a lesson which I have never forgotten. He was sixty years of age, and enjoyed excellent health. Two of his brothers had died of a sudden death, and in one of them the cause was found to be rupture of an aneurism.

For some years past, this gentleman complained of a violent pain about the base of his chest, in the course of the intercostal nerves; the pain was most intense in front, and where it was so, the skin was also slightly benumbed. It sometimes left the chest, and spread to the sides of the neck and head, where it simulated a neuralgia.

The symptoms were not constant, but returned at uncertain intervals. All the medical men whom the patient had consulted, and I among the rest, thought that the case was one of rheumatic neuralgia. After a few years, the pain became almost continuous, although it was very bearable. When the patient tried to walk, however, it became so fearfully intensified that he was compelled to remain almost motionless. Rest made everything right, as is the case in angina pectoris; but he often could find no relief except by lying flat on his stomach on a couch. He tried I know not how many plans of treatment. His great wealth allowed him to consult the most eminent practitioners, and to spend two or three months every year at various mineral springs. At last, he complained to me one day of a queer throbbing sensation in the back, on a level with the seventh and eighth ribs on the left side. On laying my hand over that part, I felt an impulse isochronous with the heart's beat. From that time, percussion and auscultation settled all doubt about the existence of aneurism of the aorta. The disease made rapid progress; four ribs became eroded after a time, and a tumour of the size of a child's head showed itself under the skin. I need not add that the case terminated as such cases always do; the aneurism destroyed the skin, and burst suddenly outside.

A few years ago, I saw with my colleague, Mr. Richet, a merchant who was exactly in the same state. He complained of pain in the base of the chest, which recurred in paroxysms, and if, instead of following the course of the intercostal nerves, this pain had been seated in the nerves which are usually affected in angina pectoris, it would have been confounded with this affection. It was for a long time ascribed to rheumatism, and the most varied and the most energetic treatment was vainly

tried. At last, after several years had elapsed, stethoscopic examinations, which until then had revealed nothing, enabled us to recognise the existence of an aneurism of the thoracic aorta. I at once foresaw the issue of the case, and, indeed, death occurred suddenly a few months afterwards, during the night. The patient was residing at the time at Saint-Germain-en-Laye, and my excellent friend, Dr. Lepiez, who made the autopsy, ascertained that the aneurism had burst into the pleural cavity.

The close relation between these symptomatic neuralgias, the history of which I have just related, and angina pectoris is sufficiently evident.

Besides, if we study neuralgias of other regions, we shall find that they pretty frequently take on this paroxysmal course of angina pectoris.

The frequently perfect periodicity presented by neuralgias due to some grave organic lesion is something very remarkable. I have already related to you the cases of two ladies suffering from carcinoma of the uterus whom I saw with Récamier and with my excellent friend, Dr. Lasègue. In 1862, I saw a third case of the kind, that of a lady with a uterine polypus, whom I attended with Professor Nélaton. In all three the most fearful neuralgic pain recurred every day at the same time, with the regularity of the most typical ague.

Some of you may also remember a man who was at No. 10, in St. Agnes ward, and who suffered from pains returning every day at the same time, with unspeakable violence, sometimes accompanied with an attack of unilateral eclampsia, after which there remained some hemiplegia. After death, we found cancer of the brain.

I lay so much stress on the perfectly periodic character of neuralgias, due to the gravest organic lesions, because some pathologists have asserted that periodicity, when well marked, was a character distinguishing pure neuroses from neuralgias depending on a grave organic visceral lesion.

In the case of angina pectoris, the periodic recurrence of the attacks by no means, therefore, excludes the idea of an organic affection of the heart, or its valves, or of the great vessels. I admit, and the majority of practitioners do so, that this singular neurosis may be symptomatic; but I admit it merely in this sense, namely, that there is a mere coincidence, and that the organic lesions, whatever they may be, only afford an opportunity for the development of the neurosis which is superadded to them.

I merely advert now to the fact that neurosis may be engrafted on organic lesions, and be independent of them since those lesions are persistent, and cannot, therefore, be regarded

as the essential condition and the true cause of nervous disturbances which are of a transient character. At some other time, I mean to go more deeply into that question.

What, then, are the causes of angina pectoris? I mean, of course, the *predisposing causes*, the exciting or determining causes being set aside for the time.

Fothergill relates the following case, which has been quoted by Desportes :—[1]

"A man, about thirty years old, of rather small stature, with a short neck, and of a robust constitution, and used to taking moderate and regular exercise, was subject to a complaint of such marked characters that it could not be mistaken for any other. Whenever he walked up a hill, or even whenever he walked a little faster than usual, or if, when riding, he made his horse gallop, he was obliged to stop suddenly, on account of a constricting sensation which he had in his chest, and which, he said, made him fear that he should die if he were obliged to move on. This sensation was felt across the chest, and extended along the arms as far as the elbows; it lasted a pretty long time. Moderate exercise of any kind did not give rise to the sensation. The patient had noticed that he suffered less when he moved about with an empty stomach than when he had taken food. The lungs did not seem to be affected; there had been no cough, no symptoms of inflammation, no bronchitis, no signs of hydrothorax, no transient fit of anger to account for the production of such sensations.

"Fothergill recommended light diet, the bowels to be kept properly open, moderate horse exercise, and the avoidance of long and fatiguing walks. He prescribed a few soap pills, native cinnabar made up into pills with some gum, and a weak bitter tonic with iron for a few months. The patient afterwards took the Bath waters for several seasons. He got perfectly well, and was enjoying good health twenty years afterwards, or at all events, Fothergill had not then heard that he had been attacked with the same pain again."

This case, gentlemen, is quoted as an instance of idiopathic angina pectoris. It would be difficult, indeed, to find another cause for the complaint than a strange predisposition on the part of the patient. Similar cases are not perhaps as rare as might be supposed, and you will doubtless have occasion to meet in your career with individuals who will tell you that they have had similar sensations in various degrees. These neuralgic pains which, starting from the præcordial region, and attended with a sensation of constriction of the chest, radiated to the throat, and extended to the arm, have either shown themselves once only or have been transient, and never returned, so

[1] Traité de l'Angine de Poitrine, Paris, 1811.

that they never felt sufficiently alarmed to apply to a medical man, and it is only by chance that they ever mention them. Yet, as the case which I have just quoted shows, idiopathic angina may, as regards the frequency and intensity of its paroxysms, be exactly like that which is due to more palpable causes.

Among such causes, *rheumatism* and *gout* must be ranked.

Some authors have thought that *angina pectoris* was merely a manifestation of the rheumatic or the gouty diathesis, which settles upon the heart, according to the majority, but, according to others, on the lungs and even on the stomach, the cardiac symptoms occurring then only from sympathy with the gastric disturbance. Without adopting such an exclusive opinion, I think that angina pectoris is in some instances indeed a rheumatic or gouty affection. A retrocession of gout or of rheumatism need not be appealed to, for it is conceivable that this neuralgia may develop itself in the same manner as the other neuralgias from which gouty and rheumatic subjects generally suffer.

The following cases are instances in point:—

On February 2, 1861, I was consulted by M. B. de R., a patient of Dr. Maugeret (of Tours). He was sixty years of age; his father had suffered from asthma, and he had himself all the appearance of a gouty subject, and, for the last six years, had had saccharine diabetes. He told me that shortly after he had begun to pass sugar in his urine, he had been seized with angina pectoris, which presented somewhat unusual characters. The paroxysms recurred about one o'clock in the morning, independently of difficult digestion, or, as sometimes happens, of bad dreams. It began with an acute pain in the muscles of the left arm, and radiated from there towards the chest, a little above the heart; it went on increasing progressively for an hour or two, then diminished slowly, and ceased about morning. These attacks returned several nights in succession, and after disappearing for a few days or a few weeks, they recurred again with the same characters. Although the pain was very acute, it did not prevent him from drawing in a deep breath at will, and he had never had a sensation of imminent suffocation.

During the day he could walk easily on even ground, but if the ground had the slightest ascent, if he went up a staircase pretty quickly, he was obliged to stop, under pain of being compelled to sit down or of falling. These symptoms came on if he took the least exercise after dinner, and increased a little by degrees every year; they were relieved by rubbing in belladonna ointment into the left arm-pit.

I examined his heart and great vessels with the greatest care, but detected nothing abnormal.

Now, do not these nocturnal attacks of angina pectoris remind you of fits of asthma, at least as regards the evolution of the phenomena?

Two days after I had seen the above patient, on February 4, Mr. T., a former pharmaceutical chemist, sent for me. He was suffering from capillary bronchitis, accompanied with strange pain in the chest, something like angina pectoris, which dated six months back. Thus, from time to time, for several days in succession, he could not take the least exercise without feeling a violent and sudden pain behind the middle of the sternum, with extreme difficulty of breathing. This pain extended immediately to both arms, but was more intense in the left. He felt a little relief only by stopping short, and raising both his hands to his head, when his arms immediately felt benumbed. All was over in about a minute; but the paroxysm lasted longer if the patient did not at once obey an irresistible call to pass water; and if he had four attacks in an hour, he was obliged to pass water four times. He added also that, when the paroxysm was drawing to its close, as when his arms were getting numb, he felt the mucous membrane of his nose get congested.

These very frequent and almost irresistible calls to pass water, which are likewise present in some cases of asthma, do, in my opinion, establish an analogy between this case and angina pectoris.

On July 24, of the same year, I was consulted by a Sicilian gentleman, aged forty-eight, tall and robust, whose father was deaf and dumb, and rather gouty, and whose maternal grandfather had been the subject of the most acute gout. He was habitually dyspeptic, had for many years suffered from cutaneous eruptions, and was subject to headaches. In 1858, he had had a violent attack of gout in the big toe, which he treated by leeches and colchicum, and which disappeared suddenly. In the following year, his dyspepsia became worse, and he soon afterwards had paroxysms of angina pectoris, beginning in the left arm, and extending with rapidity to the heart. The pain and sensation of thoracic constriction were so fearful that he thought he was going to die. These attacks recurred chiefly during the night, and came on during the day if he took the least exercise, rarely lasting more than three minutes. His intellect was unimpaired. After an indifferent treatment, he improved, and was well when he came to Paris; he could walk fast and go briskly up a staircase without feeling anything. I advised him not to interfere with his gout if it ever appeared again, and I enjoined, as I do to all gouty subjects, great regularity and sobriety in his manner of living, and exercise. I meant to try an appropriate treatment when the attacks

returned. The heart and great vessels seemed to me to be perfectly healthy.

I saw this patient a month afterwards, and his health was still excellent. He had not had angina pectoris. I examined again his heart and great vessels with the greatest care, but found nothing wrong with them.

Ten days before, on July 14, I had been consulted by a lady, aged fifty-five, who for the last seven or eight years had had several attacks of gout. In the beginning of the year 1862, she had had her first attacks of angina pectoris. The pain began at first in both shoulders, spread rapidly to the tongue, the neck, and then to the arms and chest. It came on if the patient made the slightest movement, or was in the least moved, and it was not accompanied by numbness. The attack seldom lasted one, two, or three minutes, and terminated more quickly when perspiration set in.

On the most careful examination, I found no sign of organic lesion of the heart or of the great vessels.

If we take such cases into account, then, in which gout, as may be at least supposed, had something to do with the occurrence of angina pectoris, it would appear that angina pectoris, like asthma and other neuroses, may be a manifestation of the gouty or the rheumatic diathesis. But there is a predisposing cause, which cannot, I think, be called in question, and which I have already mentioned, although no one has indicated it, namely, *epilepsy*. In some cases, and perhaps in a pretty good number of instances, according to my experience, angina pectoris is an expression of this cruel and fearful complaint, and is a variety of the vertiginous form of the disease, in other words, it is an epileptiform neuralgia. Its invasion is as sudden, its progress as rapid, and its disappearance as sudden, and, as I have already told you, it is not of very uncommon occurrence to find persons who have in former years suffered from angina pectoris become subject afterwards to epileptic fits, just as in other instances angina pectoris has been preceded by well-marked epileptiform seizures. A case of the kind lately again came under my notice.

A gentleman, aged forty-five, who was subject to epileptic fits, had for the last six months suffered from symptoms of which he gave me the following description. Whenever he took exercise of a somewhat violent character, or walked a long distance, he suddenly felt a painful oppression at the chest. For the last month this symptom had recurred three times a day, even when he was quiet, and it had become very severe. He had an acute pain, seated at first in the right half of the chest in front, giving rise to the sensation as if he wore a padded plastron; and after a minute it spread to the corre-

sponding arm, which felt very numb and painful, and was of a higher temperature than the left. These symptoms lasted for a quarter of an hour nearly, and then disappeared entirely.

At the outset, there was an abundant secretion of intestinal gases.

The patient's general health seemed to be excellent. His appetite was good, his digestion regular. He complained of nothing besides the symptoms for which he had come to consult me, and I found no symptom, no sign of organic lesions of the lungs or of the heart and great vessels. I recommended a treatment by belladonna and bicarbonate of soda, of which I shall speak by and by.

In addition to the suddenness of its access, the rapidity of its progress, and its abrupt cessation, angina pectoris presents, in other respects, many points of resemblance to epileptiform neuralgia. When I spoke to you of this latter complaint, I told you that the pain, which constitutes its chief element, is accompanied with congestion of the affected region. The same thing occurs in angina pectoris, as in two cases which I have related to you, and as I shall take care to point out when I come to the description of the symptoms.

I do not think that it has been proved that males are more subject than females to this singular affection.* It is certain, however, that angina pectoris almost exclusively attacks individuals above forty or fifty years of age, although it has been met with in young persons. Fothergill's patient was about thirty years old, and Desportes has related the history of an individual, aged twenty-five, in whom, I may add in passing, "the lungs, heart, coronary arteries, great thoracic vessels, or the valves at their cardiac ends, disclosed on examination after death no change whatever, no induration, no ossification." Heberden had also stated that angina pectoris could come on in youth, and Robert Hamilton, that it did not even spare childhood.

Since this neuralgic affection may be the expression of a diathesis, we need not be surprised that the fact has been admitted that it may be *hereditary*. Hamilton relates that a soldier, who had angina pectoris, assured him that it was an hereditary complaint in his family, and that his father, his two brothers, and his sister had suffered from it.

As is the case with all neuroses, the *exciting causes* of angina pectoris are exceedingly numerous and variable. The patient is often seized with a more or less violent paroxysm, without being able to know the reason why; he may be even seized during sleep. This especially occurs when the angina pectoris is merely an epileptic aura.

[1] [Out of eighty-eight cases of angina pectoris collected by Sir John Forbes, only eight occurred in females.—Ed.]

Some patients state that sudden atmospheric changes bring on the paroxysms, or that they cannot walk, run, or ride, against the wind without being compelled to stop from an attack of the complaint.

The most frequent causes, especially when the angina pectoris is due to an organic lesion of the heart or of the great vessels, are sudden movements, unusually active exercise, as brisk walking, or the act of going up a staircase, or, again, fits of coughing, prolonged speaking, straining at stool. These efforts or these muscular movements need not even be very violent, since, as in the case of the military superintendent whose history I related to you in the beginning of this lecture, the pain came on after the patient had been engaged in putting his signature to many papers.

In some instances, the first seizures come on after some excess in eating or drinking; in many cases, the paroxysms are always more violent after a meal, even when moderate, whether the individual moves about or remains quiet. Jurine has, however, recorded the case of a man whose attacks were most violent and prolonged when he was fasting.

Deep mental emotions, especially fits of anger, are frequent exciting causes of angina pectoris, and they not only bring on a paroxysm, but they increase the intensity of the disease to such a degree as even to cause death.

Such are the circumstances in which the singular affection which has engaged our attention to-day comes on in the majority of instances, although no rule can be laid down concerning it. Their multiplicity shows the essentially nervous nature of the complaint, and this fact will become still more evident from the changeableness of the symptoms.

It almost never happens that angina pectoris is ushered in by premonitory symptoms: its access is sudden. Pain is suddenly felt behind the sternum, accompanied with a sense of constriction and anxiety, generally seated in the left side of the chest, but occasionally in the right side, and it is so intense as to make the patient dread suffocation and syncope, and to deprive him of the power of speech.

It is rarely confined to that part, for in nearly every instance it spreads simultaneously, sometimes along the neck as far as the articulation of the lower jaw, the movements of which are impeded, but more frequently along the pectorales muscles to the shoulder-joint, from which it descends along the inner aspect of the arm as far as the elbow, and down the fore-arm to the fingers.

The left side is the one generally attacked, as I have told you, but in some cases the right is the side affected, as in the epileptic patient whose history I have related to you. In other

cases, instead of ascending to the neck or arm, the pain descends to the epigastrium as far as the groin; in others, again, but very rarely, it is felt in all those regions at the same time.

Its extension to the upper extremity is so constant a phenomenon that some authors, particularly Wall, who described angina pectoris nearly simultaneously with Heberden, have given it as an essential character of the disease.

It has occasionally been known to follow an opposite course, beginning in the arm, and thence quickly spreading to the chest. Do you not find, gentlemen, great analogy between this and what occurs in the *aura epileptica*, and is not this in contradiction to the view that angina pectoris is of necessity caused by a material lesion of the organs contained in the thoracic cavity.

Sometimes, again, this pain is felt in the hand alone, without starting from the chest, and without passing along the nerves of the arm, or taking an ascending course.

On March 29, 1863, I saw, in consultation with Drs. Gruby and Maître, a Russian nobleman suffering from hypertrophy of the heart with systolic bellows-murmur at the apex. He felt from time to time an acute pain in the cardiac region, which disappeared after having been strictly local; then all of a sudden, without any manifestation about the heart, he had in the left hand a pain which he compared to that of cramp, and which was accompanied with numbness. There was no muscular spasm. The pain lasted about a minute, and disappeared without leaving any traces.

Lastly, in some cases, angina pectoris consists in violent palpitation, with numbness of the left arm, without pain. This was the case in a young married lady, aged twenty-two, who consulted me on November 22, 1862. Her grandfather had been gouty, her mother suffered from violent neuralgias, and she herself had been subject to angina pectoris since she was sixteen years old. For the space of four years, she had only had excessively violent palpitation, without any sensation in the arm, but for the last four years the palpitation was accompanied by painless numbness of the left arm, which compelled her to drop whatever she might be holding in her hand. These symptoms recurred whenever she took a little more active exercise than usual. I found no signs of cardiac or valvular lesions.

When pain is present, as unquestionably happens in the great majority of cases, it is not generally increased by making pressure over the affected parts, or by moving the arm into which it extends. Nay, mere pressure may relieve it, and I may again remind you on this point of the patient, whose history I have already related to you, and who used to relieve his

throbbing pain by squeezing his back against a piece of furniture.

Although patients suffering from angina pectoris think they are going to be suffocated during a paroxysm, the chest is normally resonant on percussion, and if it be ausculted as they draw in breath again, vesicular breathing is heard everywhere. This is far from being the case in fits of dyspnœa.

Should the patient assume any peculiar attitude, it is on account of the pain, and not from distress of breathing. Very varied attitudes are assumed: one patient will lie motionless on his back; another will incline backwards on the back of his chair, or on his pillows; a third will place himself on all fours, resting on his knees and elbows; while a fourth may stoop so much as to bend in two.

During a paroxysm, the face turns pale, and soon afterwards becomes more or less red; this congestion, which I have compared to what occurs in epileptic fits, occurs also in other parts which are painful. Thus, in the woman in St. Bernard ward, I noted that the skin of the left hand, which was very painful, became at first extremely pale, and then of a livid bluish hue. Sometimes, also, the face and limbs are bedewed with perspiration.

The intellect is, in general, unimpaired all the time, although some exceptional instances have been recorded of individuals who had a wandering look, and who muttered unintelligible words, as if in a state of ecstacy. Lastly, there is sometimes loss of consciousness, as the pain goes off, especially if the angina pectoris be an aura epileptica, although the intensity of the pain, and perhaps some great disturbance in the heart's action, may bring on syncope of a very different character from the loss of consciousness which accompanies the epileptic form of angina pectoris.

When the disease manifests itself for the first time, the paroxysms are transient, and last scarcely a minute or two; but when it is of old date, the attacks may last several hours, and even several days, with exacerbations.

The attack terminates as suddenly as it began, but the patient retains for some little time longer a sensation of numbness in the regions which have been the seat of pain. If the paroxysms have been frequent and violent, from the disease having reached a great degree of intensity, there remains for a longer or shorter period trembling and weakness either of the whole body or of the affected limb only, which may persist until another paroxysm takes place. An individual may have only one attack of angina pectoris, and be rid of it for ever. Such cases are very rare, and it is doubtful whether the diagnosis was then very accurate. In the majority of instances, several paroxysms

follow one another, at more or less distant intervals, after years, twelve, six, or three months, or weeks, the intervals becoming shorter in proportion as the lesion which gives rise to this complaint makes progress. We have seen that the paroxysms may return periodically. In the intervals, the person apparently enjoys perfect health, unless, of course, the angina pectoris be due to the presence of an organic affection, as some disease of the heart or great vessels, to which the general condition is subordinate. From what I have told you of its coexisting with organic lesions, in perhaps the majority of instances, and of its being one of the manifestations of the vertiginous form of epilepsy, it evidently follows that angina pectoris is a most serious complaint, as being a symptom of diseases which sooner or later terminate in death. Although, from its nature, idiopathic, rheumatic, or gouty angina pectoris admits of a less severe *prognosis*, this should in all cases be extremely reserved. For there are occasional though rare cases on record of individuals who have died during a paroxysm, and I may mention, among other instances, that of John Hunter, who died suddenly after a fit of anger which caused the recurrence of angina pectoris to which he had been subject for eight years.

The disease may terminate fatally shortly after the manifestation of the first paroxysms, or the patient may live for many years, whether the attacks recur at nearer intervals and increase in intensity, as is generally the case when the angina pectoris is symptomatic of a cardiac affection, or is the expression of epilepsy, or whether they recur at distant intervals only, decreasing in intensity or persisting to a less degree.

The disease is curable when it is not under the dependence of an appreciable cause, or when it is due to a rheumatic or gouty diathesis; and this happy result may be especially expected when the patient is young, and still more when the seizures have been of moderate intensity. The complaint is almost unavoidably fatal when it is hereditary.

The violence of the seizures, the facility with which they recur under the influence of exciting causes, necessarily increase the gravity of the prognosis; and hence may be inferred an important rule for treatment, namely, that these causes should be avoided, and, above all, mental emotions, which are perhaps the most powerful of all.

The extreme variability of the phenomena which characterise angina pectoris often render its *diagnosis* very uncertain, and it is not surprising that very different conditions have been confounded with it. As Wichmann remarked, twenty-five years after Heberden, an individual need only complain of anxiety, and of a sense of constriction about the chest, even of impeded breathing, for its being immediately ascribed to angina pectoris.

Thus, pleurodynia of the præcordial region, which set in suddenly, and temporarily impeded respiration, disappearing rapidly, has been mistaken for angina pectoris. The pain, in such cases, is more superficial than that of angina pectoris, and does not, like it, shoot beyond the part which it attacked in the first instance. It is seated in the pectorales muscles, and is relieved and even removed by taking in a deep breath, prolonged for a while, and by making pressure on the affected part. Lastly, it is not lancinating, and is not accompanied by a sense of anxiety, and is not followed by a feeling of numbness like angina pectoris.

Thoracic and cervico-brachial neuralgias are distinguishable from angina pectoris by the pain being felt only in the course of the diseased nerves, and by its being continuous, although it recurs in paroxysms, and by its obstinate persistence for a more or less prolonged period. Its accession and disappearance have not, therefore, the suddenness which characterises angina pectoris.

The diagnosis is made with difficulty when an individual afflicted with aneurism of the aorta suffers from sternal pains, shooting towards the shoulder, and accompanied by a sense of choking which, from its growing worse at times, might lead one into error. But, even then, these pains do not recur in very distinct paroxysms; they are continuous, or, at least, never cease spontaneously. The same remark applies to the pungent, lancinating, and excruciating pain, attended with oppression at the chest, which occasionally supervenes in pericarditis.

In conclusion, in spite of their extreme diversity, the characters of angina pectoris are such that it seems to me difficult to mistake them.

As a rule, I know nothing so difficult as the treatment of nervous disorders. Neuroses are not only capricious in respect of their etiological conditions and of their symptomatic manifestations, but of their amenability to treatment also. Some patients get well after the use of remedies which fail in others, and a treatment which has proved unsuccessful in one case is sometimes followed by the best results in instances apparently perfectly similar. The very variability of their manifestations, the suddenness of their invasion without any appreciable cause, and their oft-unexpected abrupt cessation frequently throw doubt on the real utility of our interference. This is especially the case in angina pectoris.

The paroxysms are often of such short duration, and generally terminate so suddenly, that their disappearance can hardly be ascribed to the influence of treatment. If they have been brought on by somewhat active exercise, as a brisk walk, or by running, the patient need only stop still to cause the pheno-

mena to pass off, although cases have been recorded in which individuals have, in defiance of the pain they felt, continued to walk, and have got rid of it. Some persons have been able to stop the paroxysm by forcibly holding their breath. I have already alluded to Jurine's patient, who had most violent seizures when fasting, which recurred in great frequency unless he immediately took some food.

Although we can with difficulty appreciate in a just measure the utility of a particular remedy, we can judge of the opportunity of certain modes of treatment by taking into consideration the nature of the symptoms which we are anxious to remove. Now, to begin with, there is a measure which I must distinctly reject as inapplicable to a complaint like angina pectoris, in which there is such an imminent risk of syncope, I mean *bloodletting*. Although it has been recommended by eminent physicians, and even by Laennec, it seems to me to be, to say the least, irrational, whether the blood be removed by opening a vein at the elbow, or by applying leeches to the epigastrium or to the præcordial region.

Emetics, antimony, in particular, have been vaunted in the violent seizures, but they seem to me to be contra-indicated, on account of their lowering influence on the system.

Diffusible stimulants, ethereal preparations, ammonia in small doses, alcoholic infusions of mint, are much more indicated during a seizure than opium and other narcotics which have been lauded as so beneficial.

When the paroxysm is prolonged, and there is a marked tendency to syncope, the effects of these remedies taken internally may be aided by the use of stimulating alcoholic or ammoniacal liniments, by dipping the hands or feet in hot water in which mustard has been dissolved.

But we should particularly endeavour to avert the paroxysms, and not combat them when they recur.

A great many methods of treatment have been in turn adopted and laid aside, some of which were altogether empirical, and others founded on the various opinions held with regard to the nature of angina pectoris. Narcotics, *opium*, or its active principles, solanaceous preparations, lactuca virosa (the latter vaunted by Schelinger, of Frankfort) headed the list.

I recollect a patient afflicted with very severe angina pectoris, recurring in paroxysms several times a day with alarming violence, who improved rapidly, and obtained what he termed *a cure* by the use of frictions, made several times a day, over the sternum with a liniment of stramonium. Hypodermic injections of atropia, about the starting-point of the pain, and in the neck and arm-pit, in some cases retard the seizures, diminish

their violence, and ultimately cure, especially if the neuralgia be not dependent upon an organic affection of the heart or aorta.

On the hypothesis, admitted by some, as I have told you, that angina pectoris is due to ossification of the coronary arteries, *phosphoric acid* has been recommended, with the view of preventing and even of removing these ossifications. I need not add that this absurd idea could only occur to a chemist, who should have studied physiology and medicine before dabbling in therapeutics.

Bretonneau, whose practical sense had not been stultified by the most extensive chemical knowledge, and who in proportion as he grew older in practice openly confessed the deplorable errors which chemistry had led him to commit, and the little assistance he had derived from it in therapeutics, Bretonneau was yet led by a chemical theory to adopt a useful treatment in angina pectoris.

Although he was successful, I have often heard him laugh at the theory which he had imagined, and express surprise that, for once in his life, chemistry, the favourite science of his youth, had helped him to do some good in therapeutics.

This is the manner in which the illustrious Tours physician was led, as he stated himself, to adopt his peculiar treatment of angina pectoris. He, of course, thought that the complaint was due to calcareous concretions of the first part of the aorta. "On being consulted by a person afflicted with angina pectoris, I asked myself whether he would not derive some benefit from the prolonged use of *bicarbonate of soda*, from which such marvellous results are often obtained in the calculous diathesis. There was such a great difference, however, between the concretions proper to angina pectoris and urinary calculi that it was very doubtful whether any good results would be obtained, even by persevering with the greatest docility and patience in a treatment based on such vague reasons. I felt, therefore, more pained than surprised when, after this treatment had been tried for two months, I ascertained that no favourable change had followed.

"Yet, from that time, it became evident that, if the complaint had not yielded, it had not been aggravated; then it manifestly improved, and after the patient had made use for six months of the artificial Vichy water, he got rid entirely of all his symptoms of angina pectoris."

Thus, happily helped by chance, Bretonneau often repeated the experiment after that time, and treated several cases successfully. The bicarbonate of soda is given after a peculiar method. First in doses of two scruples; one scruple before each of the two principal meals; and this quantity is to be gradually increased, if it be well borne by the patient, to eight

and even ten scruples a day, the patient taking from two to two and a half scruples half an hour before and immediately after each of the two principal meals in the day. Bretonneau recommended to increase the dose gradually for ten days, and then by degrees to diminish it for the next ten days. The treatment is then suspended for the space of fifteen or twenty days, after which it is resumed, and is continued in the same manner for more than a year, to be again followed after an interruption of several months.

Bretonneau combined belladonna with the carbonate of soda, and administered it according to certain rules also. He prescribed pills, consisting of one-tenth of a grain each of extract and of powdered root of belladonna.

The patient takes in the beginning one of these pills in the morning, a quarter of an hour before his first meal, and does so for three days running. For the next ten days, he takes two at the same time in the day, and at once. For twenty days, three, always at once. If no improvement follows, the dose is increased to four pills, and should the paroxysms recur with the same violence and frequency, the dose of the medicine is increased by one-fifth of a grain every ten days, unless there supervene unpleasant dryness of the mouth, marked disturbance of vision, accompanied by a very striking dilatation of the pupils, showing that the too rapid increase of the dose has produced effects which should be guarded against. Whenever, therefore, a progressive amelioration has been obtained before this rapid increase in the dose of the medicine has commenced, it should not be increased again; and it is only when the improvement obtained seems to diminish that the daily quantity administered should be raised by one-fifth of a grain.

Belladonna should be persevered in during the intervals when the bicarbonate of soda is stopped.

This treatment is, of course, beneficial in those cases only when there is no vascular lesion, precisely in cases the very opposite of those suggested to Bretonneau by his chemical theory.

This treatment of angina pectoris by belladonna is, as you may see, exactly the same as the one which I recommend in epilepsy. And you need not be surprised at this, for, as I have told you, angina pectoris, in many cases, is only an epileptiform neuralgia, or a kind of aura epileptica.

For the same reason, you will understand how cases have been recorded in which angina pectoris had been successfully treated by *nitrate of silver*, which has also been lauded in epilepsy.

I shall not enumerate to you all the remedies that have been vaunted in this complaint, but will merely mention that

Alexander, quoted by Harles in his monograph on the use of *arsenic* in medicine, has related the history of a man, aged fifty-seven, who got rid of a very severe angina pectoris by taking six drops three times a day of Fowler's solution.

I do not dwell on the hygienic rules to follow, because it is above all things clear that the patient must avoid all causes capable of bringing on a paroxysm. That he should take exercise in moderation, should observe perfect rest of mind, and avoid all deep mental emotion, are precepts the necessity of which is self-evident.

I will not bring this lecture to a close without speaking of the use of electricity, which holds a very important place in the treatment of angina pectoris. We are indebted to Dr. Duchenne (of Boulogne) for the methodical employment of this therapeutic agent, which is sometimes so powerful.[1]

I will, with your permission, read to you a case published by Dr. Duchenne. It affords another proof that the most violent angina pectoris may not be due to an organic lesion of the heart or the great vessels, because, had such a lesion been present, electricity might have relieved the pain, but could never have cured the patient, particularly in such a short time.

"Pérone, aged fifty, a currier, residing at Belleville, 25 Tourtil Street, of a stout build and sanguine temperament, rather fat and with a short neck. He has never had any serious illness. Two years ago, he had some rheumatic pains in the right shoulder, which compelled him to interrupt his work for a month, although he had no fever. He is not generally afflicted with short breath, and is not subject to palpitation. He lives in a healthy place, and his apartments are not damp.

"On November 29, 1852, at nine o'clock in the morning, before breakfast, he suddenly felt, without any known cause, a deep burning sensation in the upper and middle regions of the chest, and a pain which extended into the left upper extremity.

"He had at the same time a sensation of tingling, which went on increasing from the elbow as far as the tips of the fingers. During this attack, his heart beat with force and rapidity, his head felt heavy, was rather painful, and he spoke with difficulty from insufficient respiration, and the attempt increased the pain. He was compelled to stoop forward, to keep still or to sit down, as his pain was worse when he held himself erect; he felt extreme anxiety, and had a dread of impending death. This first attack began to diminish only eighteen hours after it had set in, after a copious bleeding. Mustard foot-baths, sedatives taken

[1] De l'électrisation localisée et de son application à la pathologie et à la thérapeutique; 2nd edition, Paris, 1861.

internally, and a warm bath had been tried at first, but without any good results. The improvement obtained was not very great, because the patient was compelled to observe the most complete rest, in the sitting posture, as a fresh paroxysm followed any attempt at lying down. The seizures recurred for the slightest cause; sneezing, yawning, or the least emotion, was sufficient to bring one on. During the day, he was perfectly calm, except when he had a paroxysm, lasting about eight or ten minutes, as severe as the former ones, and brought on by his attempting to move, or by any emotion. He could not sleep. By degrees the seizures became less frequent, although they continued as violent as before, and frightened both the patient and those about him.

"There was no disturbance of the appetite and of digestion; and there never was any fever during the course of the disease. A fortnight after the accession of the complaint, frictions with tartar emetic ointment were made over the front of the chest; a purgative was administered every fourth day, and twenty leeches applied to the arms. In spite of this treatment, the paroxysms returned whenever the patient took the least exercise, so that he was compelled to remain perfectly quiet. Dr. Mongeal, his medical attendant, on seeing this condition continue, decided on sending the patient to me, with the idea that contraction of the diaphragm might be at the bottom of the complaint, which he justly termed *angina pectoris*, in the letter which he wrote to me.

"I noted the following circumstances when Pérone consulted me on April 28, 1853.

"He rode from Belleville to my house, and when coming up to my apartment, which is on the second floor, he was obliged to stop on every stair, on account of a sense of constriction in his chest and of the other phenomena which I have described above. He became perfectly quiet after resting for a quarter of an hour; on auscultation and percussion, nothing wrong was found with the bronchi, lungs, heart, or great vessels: the pulse was normal. Pressure made over different parts of the chest gave no pain.

"I then asked him to bring on a paroxysm, which he could do by stooping as if he wanted to pick up something. The following phenomena then manifested themselves simultaneously: a very acute deep burning pain, with sensation of constriction, was set up in the upper part of the sternum, shooting into the left upper extremity, running along the posterior aspect of the arm, and the outer of the fore-arm, and terminating in the index finger; numbness and formication were felt in the whole of the limb. The patient held all the time both his hands folded on the upper part of the chest, compressing it as if to

relieve the pain. His head was bent forwards, his shoulders drawn upwards and forwards through the contraction of the pectoralis major and part of the trapezius: the pain became worse whenever he tried to stand erect, or put his shoulders back. On my asking him to walk, he had no sooner taken a couple of steps than he was obliged to stop and sit down, on account of the increased intensity of the pain behind his sternum. His breathing was short and agitated, his heart beat violently, his pulse was frequent, his face red and injected, his eyes opened wide, his body covered with a profuse clammy sweat, and his physiognomy expressive of extreme anxiety. His respiratory sounds were perfectly pure, however, and the valvular clicks well marked. The heart was of normal size, and percussion of the thoracic walls disclosed no abnormal dulness.

"When he tried to speak, his voice was broken and weak, and came out with difficulty, and his pain was increased.

"There was perfect isochronism between the movements of the chest-walls and of the abdomen during respiration; no pain was felt at the base of the chest, and there was no impairment of the voluntary movements; there was only numbness of the left arm and hand, the movements of which were weaker.

"After resting for eight or ten minutes, he became calm again, but it was only by degrees that the pain and constriction of the chest disappeared.

"*Report of the experiments and their results.*—I brought on a second paroxysm by making Pérone walk, and I applied to his nipple the extremity of my induction-apparatus graduated to maximum intensity and working with very rapid intermissions. As the nipple was galvanised, he uttered such a loud shriek that I had to interrupt the current. The pain had been excruciating, but merely instantaneous, and to my great surprise, after the artificial pain which I had brought on, the pain of the angina also disappeared completely, as well as the sensations of numbness and formication which accompanied it; respiration had become quiet again; in a word, the patient felt at once in his normal condition.

"The question arose whether this sudden transition was the result of a mere coincidence, or whether it was due to the immense and instantaneous perturbation produced by galvanisation of the nipple. In order to determine the point, I had only to repeat the experiment. But it was not so easy as before to bring on a fresh paroxysm, for the patient had to go through various movements for four or five minutes, in order to do this, while, before the galvanisation, he had merely to stoop.

"The second experiment was as rapidly successful as the first; but instead of acting on the nipple, I this time galvanised

the skin of the painful part (the upper region of the sternum). I took a sort of pleasure in thus arresting a complaint, hitherto held to be beyond relief during a paroxysm, and repeated the experiment several times with the same success, and I noticed that the more I repeated it the greater was the difficulty experienced by the patient in bringing on a paroxysm, so much so that he was only able to effect it on the last occasion by rapidly walking from the bottom of the staircase to my second floor.

"On the next day, he informed me that he had been able to go back to Belleville, without feeling the least uneasiness, or having to stop, and that for the first time since the accession of his complaint, he had been able to sleep. In the morning only, he had had a sensation of constriction, unattended with pain, in the upper part of the chest; he came to me from Belleville on foot, and walked upstairs to my room without stopping or feeling the least uneasiness. In a word, he thought that he was cured.

"I suggested to him to bring back a paroxysm of angina so as to go through the same process as on the previous day. He began at once, but it was only after nearly a quarter of an hour and after making violent efforts, as when he is engaged in currying leather, that he succeeded in bringing on a paroxysm, almost as violent as the former ones. But in two or three seconds, this was again arrested by faradisation of the skin.

"From that day the post-sternal pain, the sensations of formication and numbness in the left upper limb, disappeared, and could never be brought on again. A sense of oppression was only brought on at such times, a sort of pressure over that part of the chest which was previously painful. Faradisation of the skin, practised four or five times at pretty distant intervals, removed these last symptoms, and a fortnight after the treatment had been commenced, I could allow Pérone to resume his occupation as a currier.

"A year has now elapsed since he has resumed his laborious occupation, but his angina has not returned."

Another case, which adds value to the above, was communicated to me by Aran, and I will give you the principal features of it:—

Mrs. X., aged thirty-two, of middle stature, stated that ten years previously she fell into a kind of lethargic state, which lasted seven days, in consequence of intense grief on losing one of her children. (During that time she was known to continue to breathe by holding a looking-glass before her mouth.)

This condition came to an end on her shedding tears abundantly, but for seven months afterwards, she suffered from palpitation of the heart, with extreme anxiety, difficulty of

breathing, and impairment of the intellect. She had got better, in spite of the persistent palpitation, when two years ago (in 1851) deep grief, in consequence of some reverses of fortune, brought on a new series of morbid phenomena, differing from the previous one, with regard to their characters, their course and intensity. Thus, her new complaint came on in more or less frequent paroxysms, in the intervals of which she was free from it.

These were the chief symptoms of the paroxysm: acute præcordial pain, compared by the patient to burning heat; very marked post-sternal constriction, with pain shooting into the left arm, and attended with numbness, which lasted for some time after the attack, and with complete paralysis of the limb; extreme anxiety and a terrified aspect. Contraction of the pectorales and of the muscles which flex the head forwards; exaggeration of the pain on making the least attempt to hold up the head and draw back the shoulders, no dyspnœa, as during a fit of asthma, but short and frequent breathing. The attacks were not accompanied by hysterical symptoms: thus, there was no sense of contriction of the throat, no tears, although these could be easily brought on by talking to her of her dead child, and then she loses her reason. Lastly, on auscultation and percussion, no lesion can be made out in the lungs, the bronchi, the heart, or the great blood-vessels.

Dr. Aran had been for a long time trying to cure this condition, but without success, when Dr. Duchenne told him of the important case which I have related to you. As you may imagine, so distinguished a physician as Aran did not allow the opportunity to pass of testing a method of treatment which had answered so well in an analogous case, especially as his patient was in danger of her life. Faradisation of the skin was had recourse to during the paroxysms, and the result was as happy and immediate as in Dr. Duchenne's case, so much so indeed that she was almost completely cured of her angina pectoris, and was enabled to resume her ordinary occupation.

LECTURE XXI.

ASTHMA.

Its characters differ according to the individual affected, and according to his age.—A peculiar coryza may be the only manifestation of the disease.—The same holds good with the catarrh, which is usually one of the elements of asthma, and comes on at the close of the fit, but may in some cases be exclusively predominant when it then presents peculiar characters.—Exciting causes of asthma; they are often absent; when present, they are exceedingly variable, and occasionally very singular.—Influence of external circumstances: climate, seasons, and temperature.—Opinion of authors on the nature of asthma.—Dyspnœa symptomatic of an affection of the heart or of the great vessels, of pulmonary emphysema, of bronchial catarrh.—Asthma is a neurosis, and the manifestation of a diathetic condition, gout, rheumatism, &c.—It is also a manifestation of the tubercular diathesis.—Treatment.

Gentlemen,—Through a fortuitous combination of circumstances, which will sometimes occur in an inexplicable manner, we have had at the same time in our wards several individuals suffering from *idiopathic asthma*, a disease which is common enough, but is rarely seen in hospitals. As the epithet *idiopathic* indicates, this complaint occurs independently of all demonstrable organic lesion, in paroxysms of dyspnœa and oppression, which recur at more or less regular, more or less distant periods, in the intervals between which the respiratory functions generally recover their usual regularity.

Thus, an individual in perfect health goes to bed feeling as well as usual, and drops off quietly to sleep, but after an hour or two, he is suddenly awakened by a most distressing attack of dyspnœa. He feels as though his chest were constricted and compressed, and has a sense of considerable distress; he breathes with difficulty, and his inspiration is accompanied by a laryngo-tracheal whistling sound. The dyspnœa and sense of anxiety increasing, he sits up, rests on his hands, with his arms put back while his face is turgid, occasionally livid, red, or bluish, his eyes prominent, and his skin bedewed with perspiration. He is soon obliged to jump off his bed, and if the room in which he sleeps be not very lofty, he hastens to throw his window open in search of air. Fresh air, playing freely about, relieves him. Yet the fit lasts one or two hours or more, and then terminates. The face recovers its natural complexion, and ceases to be turgid. The urine, which was at

first clear and was passed rather frequently, now diminishes in quantity, becomes redder, and sometimes deposits a sediment. At last, the patient lies down and again falls off to sleep. On the next day, he transacts business and leads his ordinary life, often having a mere recollection of his past sufferings. In some cases, however, he continues to feel a more or less undefined sensation of thoracic constriction, which is liable to be increased by movements of the trunk, and to then render breathing more laborious and difficult. In other cases, the patient complains of flatulent distension of the stomach after eating, and of an unusual tendency to doze. In the evening, about the same time nearly, a fresh paroxysm comes on, perfectly similar to the one of the previous day, which, like it, yields to recur on the following night, and again for three, four, five, ten, twenty, and even thirty nights. These paroxysms constitute a true *fit of asthma*, which sometimes terminates in slight bronchial catarrh, which in its turn disappears spontaneously, after some time. The return of the attack is not under the dependence of any law, and in some cases it takes place after the lapse of four or five years, but in others, every year and even oftener.

You will meet with individuals who suffer from nearly constant fits for several weeks or months. During the day, somewhat more active exercise than usual, a slightly brisker walk, mental emotion, some annoyance, will bring on difficulty of breathing, amounting almost to suffocation, and a disastrous sense of anxiety. In the evening, the paroxysms return regularly with greater or less intensity, without being brought on by any cause. During the night, the dyspnœa is so great that the unfortunate patient is unable to lie down on his back or on his side, and is obliged to sleep in the most varied and sometimes the queerest attitudes. Sometimes, he can only find sleep by kneeling on his bed and resting his head on his knees, or by spending the night in an arm-chair, or by propping himself up in bed in the sitting posture; sometimes again, he can only sleep standing, resting on a piece of furniture or on the mantelpiece.

Whether an asthmatic individual be in bed or up and about, he is generally seized during the night, and, as a rule, also in the early part of the night. There are, however, exceptions to this general rule, for asthma, like all nervous disorders, is capricious, and affects very marked individualities. In every case, the attack returns at the same hour generally, but this may be in the second and not in the first half of the night.

Thus, I have myself been long subject to asthma, and my fits used to return about three o'clock in the morning. I was

then invariably awakened by a sense of oppression, and I heard my clock strike three.

In some cases, the attacks are *diurnal* instead of *nocturnal*.

My mother, from whom I no doubt inherited my asthma, used to be seized between six and eight o'clock in the morning. During the rest of the day, she was as active as possible, and she had good nights.

The master-tailor of a regiment of carbineers, then stationed at Saumur, whom I knew, was regularly seized at three o'clock in the afternoon. His attacks recurred so punctually at the same time that, on account of the period of the day at which they occurred, I ascribed them to paludal influences and diagnosed larvated intermittent fever. I gave him quinine, but without any good results.

Many instances could doubtless be found of diurnal asthma, similar to the above; but they are only exceptions, and by no means invalidate the law that the paroxysms generally return at night.

In some cases, instead of manifesting itself at once by fits of oppression at the *chest*, this singular malady sets in with *coryza*. All at once, and often without his having been exposed to any of the causes which generally bring on a cold in the head, the patient begins to sneeze with extreme violence, and in the most strangely obstinate manner. His nose runs profusely; his eyes swell, and fill with tears; then, after a few hours, these symptoms disappear as rapidly as they set in, and in the course of the evening, more commonly during the night, asthma comes on with its usual characters. During four, five, or six days in succession, and even more, and nearly always at the same time, the same phenomena repeat themselves, and terminate in the same way.

In other instances, the whole paroxysm is exclusively constituted by this paroxysmal coryza, occurring independently of all appreciable cause, or under the influence of causes which are as varied and as curious as those which, as I shall tell you presently, induce an attack of genuine asthma.

At the end of January 1863, a lady consulted me on account of fits of asthma with which she was seized under singular circumstances. She lived at Narbonne, and whenever the wind blew from the sea, she had a violent cold in the head, which lasted from twenty-four to forty-eight hours, but she had no difficulty of breathing.

She added that one of her children, who was five years old, was also subject for eight or nine months of the year to coryzas, which began with endless sneezing, whenever he exposed his full face to the early rays of the sun or to a fresh breeze.

This year, again, May 19, 1863, I was consulted by an engraver,

residing in St. Martin's Street, in Paris, who for the last five years, from the month of March (when he is in the habit of going every Sunday to the country), has been subject to fits of sneezing, accompanied by lachrymation, which recurred two, three, and four times a day. This went on for two or three months, and in the interval between the attacks, his general health was not in the least disturbed. Ten years ago, he had had fits of asthma, and even now he had them every year in the month of February; these fits come on at night only. He had suffered from bleeding piles until five years ago, and his attacks of sneezing had only come on since his piles had ceased to bleed. He had never had a fit of the gout, and although he had never suffered from any skin eruption (with the exception of some pityriasis capitis), he yet had been subject every two or three months for the last five years to a kind of slight eczematous eruption, which lasted from ten to fifteen days. When this eruption showed itself, the sneezing disappeared.

I have often predicted to individuals suffering from this curious form of coryza, who had never felt anything about the chest which could justify my assertion, that they would sooner or later become subject to asthma, and they have subsequently come back and told me that my suspicions had turned out to be true.

It is a fact clinically established, however singular it may appear, and however inexplicable it may be, that, although fits of asthma come on in most instances at night, this paroxysmal coryza, which I regard as one of the manifestations of the same disease, is diurnal, and mostly occurs in the first half of the day. This was the case, as you may remember, in the instance of the man who remained a pretty long time at No. 3, in St. Agnes ward.

Asthma sometimes, again, assumes the *catarrhal form*; and then the bronchitis, which, as I told you at the commencement of this lecture, occasionally and even habitually ends the attack, seems to be the sole manifestation of the disease.

This occurs in children chiefly, although it is not of very uncommon occurrence in adults.

In the month of January 1861, I had under my care a lady, who had come from the provinces, and who was seized two or three times a year with a violent catarrh, of which I have never seen another instance. There was continued orthopnœa, with nocturnal exacerbations that were perfectly frightful, and yet the severity of the dyspnœa was by no means proportionate to the signs revealed by auscultation. Sonorous rhonchi were alone heard, and scarcely a few fine scattered mucous rhonchi: the vesicular murmur was nowhere audible. These symptoms sometimes lasted from one to two or three months

without intermission; at rare intervals only were there glimpses of improvement, which lasted a very short time, until these prolonged paroxysms ceased somewhat suddenly, leaving behind them no appreciable traces of the disturbances which they had caused.

I repeat, it is chiefly in children that this occurs. Asthma assumes such singular forms in them that it is often unrecognised. I believe I was one of the first to point out, if not its existence in young children, at least the strange forms under which it manifests itself. It is only exceptionally that they are affected exactly in the same way as adults are, and, for my part, I remember only one instance of the kind.

The patient was a Moldavian boy, aged five, who had very distinct and well characterised fits of asthma, together with some pulmonary emphysema. In his family history, there was no mention of any hereditary taint, of gout or of rheumatism. I saw him again two years afterwards; he had then a most characteristic fit of the gout, with redness, swelling, and pain in the big toe. This was the first, and has been the last, instance I have ever seen of gout at such an early age. The gouty arthritis attacked the knees, and had not the slightest resemblance to acute articular rheumatism. During this attack of gout, the boy had not a single paroxysm of asthma. The disease ran its usual course, for, as I will tell you by and by, gout and asthma are often manifestations of one and the same diathesis, and they may alternate in the same individual, as they did in my Moldavian patient.

This form of asthma, which occurs in adults, affects children only exceptionally. In the latter, the catarrhal form is the predominating one, and presents numerous varieties. I will give you cases in proof of this, which will be better than any statement.

One of my confrères, a man of a robust frame, had two children whose health was very delicate. Their mother was hysterical, but was a sensible person notwithstanding, as her sympathetic system of nerves was more affected than the nerves of the life of relation.

One of the children became one day affected with some pulmonary complaint, presenting all the symptoms of broncho-pneumonia. These came on with startling suddenness, as it were, and assumed at once an alarming aspect. I was summoned an hour after they had shown themselves. On auscultating the child's chest, I heard subcrepitant rhonchi in great abundance, and the extreme difficulty of breathing made me dread imminent suffocation. I advised a large flying blister to be applied to the whole chest immediately. Three days afterwards, the child was quite well. My treatment had been followed

by too marvellous a success, and especially had been too rapidly successful for me to ascribe to it the credit of the cure. A few months afterwards, however, the same symptoms manifested themselves, but although no active treatment was had recourse to, they disappeared after lasting forty-eight hours. This time, still more than the first, I asked myself whether we had really to do with a peripneumonic catarrh. I recalled to mind what broncho-pneumonia was in infancy. For whilst I had learnt from experience, both in hospital and in private practice, that no child dies of genuine lobar pneumonia, the disease yielding in general, if not always, without medical interference, I knew also that it was a different case with catarrhal pneumonia, and that, although a serious complaint at all ages, it was to be dreaded most in childhood, so much so, that of forty cases treated by me in hospital forty had proved fatal whatever treatment had been had recourse to. When I considered, therefore, that my confrère's son had recovered from such a fearful complaint on the first occasion in three days, and on the second in forty-eight hours, I doubted the accuracy of my diagnosis, or at least attempted to complete it by taking into account the family history. When I thought of the mother's hysteria, I inferred that, in the child's case, the nervous element must assuredly have played the principal part, if it had not been the sole agency at work. Hence, when, three months afterwards, I was again summoned to see the same boy, who, after having played as usual during the day, had about ten or eleven o'clock at night another paroxysm apparently as formidable as the previous ones, I advised that *stramonium* leaves should be burnt in his room, confining myself this time to combat the spasmodic element. On the following day, the child was up and about.

His complaint had therefore been a true pulmonary neurosis, complicated with a bronchial secretion, the presence of which had been revealed by the fine subcrepitant mucous rhonchi heard. In this it resembled many other neuroses, which, as I shall tell you on other occasions, are frequently accompanied by abnormal and exaggerated secretions. In a word, I had had to deal with fits of asthma.

This was the first time that I had seen such symptoms in a child, or rather it was the first time that I recognised their nature, because, when I appealed to my recollections, I remembered a certain number of instances which I had met with, but without understanding them. How often, gentlemen, has it not happened that very learned, intelligent, and attentive physicians have seen, without discerning them, disorders which another more careful and a better observer, perhaps more fortunate also, and better served by circumstances, has discovered and recognised afterwards! How many phenomena are

there which we vainly try to interpret, until a day comes when we are more enlightened, and perhaps, also, are better inspired, and we discover their significance! Thus, in the present instance, this was the first time that I understood a fact which I had until then misunderstood, and that I recognised asthma under this strange form which I had not yet known how to diagnose.

I know a magistrate whose wife and nieces were the strangest type of the nervous temperament. His daughter, who was subject to catarrhal affections, went to Nice to spend the winter. In the month of May, she became affected with a catarrh of such violence that her friends got alarmed and brought her back to Paris as soon as she was able to bear the fatigues of the journey. On her arrival, she was seized in the same way, and Dr. Blache and I were sent for. We found her in a state of extreme dyspnœa, and we thought that asphyxia was imminent. But remembering the case which I related to you just now, and taking into account the hereditary history of the child, I was not frightened by this apparently very grave condition, for I foresaw that this violent conflagration would be soon extinguished. We prescribed stramonium fumigations, and, in order to calm the parents' anxiety, added a mixture the effects of which were to be insignificant. Our prognosis proved accurate. Two hours after the stramonium had been used, the symptoms disappeared. The next day, the patient was well, and when we called, the friends received us with marks of gratitude and joy, doubtless ascribing to our mixture the credit of the cure. Since then, this young lady has been seized with similar symptoms several times, and they have always been quieted by fumigations with stramonium.

Taught by these cases, and my attention once aroused on this point, I never again mistook this form of asthma whenever I happened to meet it, and I have often seen it, at least in proportion to the rarity of the complaint in children. Yet few years have passed by in which I did not see one or two cases of the kind.

In the above instances, the course of the symptoms was very rapid; but you will find, in general, that they are less intense, and that they then continue for seven, eight, ten, or twelve days, especially if they are not combated in time, or actively enough. Even then, under the catarrh which overlies the nervous element so as to mask it, the essence of the disease is always the same, and its nature has not changed.

This is so true that, if you adopt in time measures capable of removing the spasmodic element, the disease yields at once and more easily than a genuine pulmonary catarrh, even if the catarrhal symptoms were more intense and apparently more formidable in the former than in the latter.

Doubtless, when the catarrhal element has been of longer duration, it is more difficult to detect the asthma, although it is even then characterised by peculiar symptoms running a strange course. There are, on the one hand, paroxysms of oppression at the chest, of suffocation, recurring intermittingly, especially during the night, and often persisting, even after the catarrh has yielded, with a violence which is not in accordance with the improvement in the inflammatory phenomena. On the other hand, the general disturbances and febrile reaction which accompany this catarrh are slightly marked, and by no means proportionate to the severity of the local manifestations.

Lastly, the paroxysms, however fearful they may be, terminate, in general, with surprising rapidity, although they recur, it is true, at more or less distant intervals. They yield to methods of treatment which are sometimes most insignificant, and it is in such cases that homœopathic practitioners obtain the marvellous good results of which they assume the credit. In very many cases, however, very active treatment is necessary in order completely to subdue the disease. Ipecacuanha, given at the onset in emetic doses, has then been in my hands of extraordinary and perfectly unexpected service, in adults as well as in children. Belladonna or atropia, followed on the ensuing days by the administration of spirits of turpentine, according to rules which I will lay down by and by, has been equally successful.

When the catarrhal element predominates, and when, as is pretty common, a slight cold caught on exposure has been the starting-point of the fit of asthma, one might be tempted to ascribe the difficulty of breathing and other phenomena to the bronchial inflammation entirely; but this would be a grave error. Without anticipating now what I shall have to tell you at length when I come to discuss the nature of asthma, I will repeat the statement which I made just now, namely, that the spasmodic element constitutes the essence of the disease. This is so little dependent upon the inflammatory catarrhal element that the same individual who may have had an attack on the occasion of a slight cold frequently has not the slightest fit of asthma if he happen to have a severe attack of bronchitis, of capillary bronchitis, or even pneumonia.

An old friend and a patient of mine, a rich capitalist, has been subject to fearful fits of asthma since the age of twenty-five. They were so continuous and so violent in 1831 that for seven months he was unable to sleep in a bed, and was obliged to sleep standing, resting the whole night against the mantel-piece in his room. In 1840, on coming out of a theatre, he caught a cold, and had an attack of broncho-pneumonia, of a very serious

character, which for a while put his life in danger. During the course of this complaint, he never had a single paroxysm of orthopnœa. Although he cannot even now sleep in a bed unless the mattresses be arranged so as to form a kind of arm-chair, he then slept flat on his back during the whole of his inflammatory attack. His colds have now peculiar characters, and make him very ill, but he never has a fit of asthma while they last.

In such cases, therefore, bronchitis plays a part in the development of asthma, but it only acts as an *exciting* cause, which finds the system in special conditions without which it could not have produced the same effects, and the latter (and this is the point on which I am anxious to lay most stress) are by no means proportionate to the former.

According to its *exciting causes*, asthma has its individualities and its fancies, as well as its peculiar modes of manifesting itself.

In the majority of cases, it comes on without any appreciable cause; in others, which are not very uncommon, the attacks are brought on by perfectly determinate causes, which vary indefinitely in different individuals, but are nearly always the same in the same individual, although their singular influence cannot be explained.

Allow me to give a few instances in illustration.

The lady, whom I mentioned just now *à propos* of coryza, told me that her mother had been asthmatic, and that she herself had been so since the age of ten, and that she never could be in a room where stalks of Indian corn were shaken without being immediately seized with a fit. She had been entirely free from asthma for five or six years, when, at the end of the year 1862, she had an attack which lasted a month, and was this time again brought on by the same cause. Whilst at Bagnères-de-Luchon, she had been suddenly seized with asthma in her bedroom, where a paillasse made of the leaves of Indian corn, on her children's bed, was being shaken.

A case was recently mentioned to me of an individual who could not pass the shop of a rope-maker without being at once seized with a fit of asthma; either the smell, or, what appears more probable to me, the dust from the flax brought on the attacks.

The worse fit of asthma which I ever had myself came on under the following circumstances.

I suspected my coachman of dishonesty, and in order to assure myself of this, I went upstairs to the loft one day, and had the oats measured in my presence. Whilst this was being done, I was all at once seized with a fit of dyspnœa and oppression at the chest so great that I had scarcely the strength to

get back to my apartment; my eye-balls protruded out of their sockets, and my pale and turgid face expressed the deepest anxiety. I had only time to pull my tie off, and to rush to the window, which I opened in search of fresh air. I am not an habitual smoker, but I then had a cigar, and took a few puffs; in eight or ten minutes the paroxysm was over.

Now what had caused this fit? Doubtless it was the dust from the oats, which were being measured, that penetrated into my bronchi. But it was unquestionable also that this dust was not enough to bring on of itself such an extraordinary attack, or the cause at least was quite out of proportion to the effect produced. I have a hundred times in the streets, or on the boulevards of Paris, or on high-roads, been exposed to an atmosphere of dust considerably thicker than the one which I had then breathed for a very short time, and yet I had never felt anything approaching to this. There must, therefore, have been something special in the cause, and it had besides acted on me whilst I was in a peculiar state. My nervous system was shaken from the influence of mental emotion caused by the idea of a theft, however trifling, committed by one of my servants, and a cause, very slight in itself, had acted on my nerves with extreme intensity.

You will find in books analogous cases.

Cullen's annotator reports that he knew a stout robust man who was seized with asthma whenever rice was thrashed in the neighbourhood of his house.

Some of you may recollect a woman who was at No. 6, in St. Bernard ward, and who had been admitted on account of rheumatic pains. She was forty-three years of age, and remarkably stout, and there was in her history, with regard to the present point, a peculiarity which struck you. Her father was living, and enjoying good health; her mother had died of dropsy, which, from her account, must have been due to a cardiac affection; she herself had enjoyed excellent health until the age of twenty-three. She married at that time, and became subject to asthma, which recurred in paroxysms at variable intervals for the space of two years, and disappeared entirely after she began to nurse her children. The fits used to come on regularly about ten or eleven o'clock at night, lasted the whole of the night and left her in a state of uneasiness and oppression, which continued till noon; from that time she was free, and went about her usual occupations. The peculiarity in the case, which you may recollect, was this: whenever she happened to be in her bed-room, when her feather bed was being shaken, she had a fit instantly, and she was never so bad as at such times. This cause of asthma has been mentioned to me in several instances.

The following are no less curious cases.

A chemist at Tours, who was slighly asthmatic, had a fit whenever powdered ipecacuanha was dispensed in his shop. It was not only when the root of this drug was powdered, the mere weighing of the powder sufficed to bring on a fearful paroxysm of dyspnœa which lasted an hour. Whenever ipecacuanha had to be dispensed, therefore, he was informed of it, and withdrew to his own apartments. No other powder, no other kind of dust, made him suffer in the same way.

I knew another chemist, of St.-Germain-en-Laye, who was all his life subject to asthma, which recurred under precisely the same circumstances as the above.

Dr. Massina published his case in the *Gazette des Hôpitaux*.

The singular effect of ipecacuanha powder was noticed and mentioned long ago. Cullen relates that the wife of an apothecary was seized with asthma whenever ipecacuanha root was powdered in her husband's surgery, even if she happened to be at the time in the innermost part of the house. Murray, if I remember aright, has recorded a similar case in his *Apparatus medicaminum*.

A chemist of the Chaussée-d'Antin stated some time ago that, when linseed or scammony, as well as ipecacuanha root, were being powdered in his laboratory, he had a violent fit of asthma, which invariably commenced with coryza.

It is not only when they are in a state of powder that certain substances produce these curious effects; their smell alone is sometimes sufficient.

Floyer[1] cites the case of a lady whose paroxysms were brought on by the least scent.

I have myself had fits of asthma if I remained a few minutes in a room where there was a bouquet of violets; and I know other people who are affected in the same way by the smell of other flowers.

Other cases might be, doubtless, added to these, if one took the trouble of looking out for them; but those which I have mentioned are sufficient to give you an idea of the variety, and especially of the curious nature, of the exciting causes of asthma.[2]

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[1] Floyer, Traité de l'Asthme.

[2] [Another very singular exciting cause of a paroxysm of asthma is that which Dr. Hyde Salter was the first to point out, namely, emanations from certain animals. In his very interesting work on asthma, Dr. Salter mentions cats and rabbits as being the only animals capable of producing such peculiar effects, but in a lecture published in the *Lancet*, for October 6, 1866, he states that "he has met with cases in which the effluvium from horses, wild beasts, guinea-pigs, cattle, dogs, hares, would immediately give rise to a paroxysm. One patient, the proprietor of a well-known equestrian establishment, always had his asthma brought on by the presence of horses; consequently he was continually

The influence exerted on the development of asthma by *atmospheric conditions*, by *climate*, *seasons*, *temperature*, &c. are no less interesting to know, and no less singular. Two years ago, a young man from Saint-Omer came to consult me. He was subject to frequent fits of asthma, and took advantage of a respite to come up to town. As soon as he got to Paris, he felt markedly better, his attacks became less violent, and after two or three days, he was nearly free from them. He seemed to me to have recovered with too marvellous a rapidity not to ascribe it to some special influence, perhaps to a change of air, and my suspicions soon turned out to be correct. The patient remained in town for three weeks, during which time he had not a single paroxysm. At last he came and took leave of me, informing me that he was going to Versailles. This trip was the test which I wanted to confirm my suspicions or not. The very first night which he spent at Versailles, at the very gates

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asthmatic. He had no suspicion of the real cause of his symptoms till he made his fortune, and retired from business, when he almost entirely lost them; but if at any time he goes back to his old haunts among the horses, his old trouble immediately shows itself."

The case of an American gentleman, given at full length by Dr. Salter, in the patient's own words, is a most remarkable instance of this strange susceptibility of certain individuals. A patient of Dr. Salter, "when a boy, was never able to keep rabbits, in consequence of the effluvium from their hutches always bringing on asthma when he went near them. Another, a lady, was always unable to visit the Zoological Gardens without being rendered asthmatic; especially was she unable to go in the animal houses. In one case, a great variety of animals, as in the American gentleman just mentioned, had the power of giving rise to asthma—horses, rabbits, sheep, oxen, and dogs; this gentleman could never go to a horse-show or dog-show without becoming immediatcly asthmatic, and being compelled to leave. Three of my (Dr. Hyde Salter's) patients belonged to a family in which this peculiarity existed for three generations, and was evidently hereditary. The grandfather was affected by cats, and could always find out by his breathing that there was one in the room. A grandson, who was also asthmatic, always had an attack brought on by the smell of guinea-pigs. A nephew could never go near horses without being rendered asthmatic, nor could he be in a room with those who had been riding. He was a country gentleman, and frequently anxious to attend agricultural meetings, but he was unable to do so from this circumstance. Another nephew told me a curious thing of himself, which, if correct, is perhaps more curious than any of the other facts. On two occasions, when staying at a friend's house in the country, he was attacked with asthma, and found in both instances that there were deer feeding at the time immediately beneath his window; on a third visit, when the deer had been removed at a distance from the house, he was quite free from any asthmatic symptoms.

"One of these gentlemen told me of a friend of his, a country clergyman, who was always rendered asthmatic by the neighbourhood of a hare or hare-skin. If he met any of his parishioners on a Sunday who had been poaching, and had their booty about them, he could always in this way detect them. When this gentleman was a boy, and studying with a private tutor, a friend put a hare under a sofa in a room where he was, as a practical joke; the result was an immediate and very severe attack of asthma."—Ed.]

of Paris, he had a most fearful attack; he did not feel as well as before on the following morning, and in the evening of that day, he had another fit. On the second day, he started for Saint-Omer, taking Paris on the way.

I had foreseen that the trip to Versailles was the test which I wanted from what the patient had himself told me. He became subject to asthma when he was nineteen years old; he was then residing in his native town; two years afterwards, he had gone to London with his father on business, and from that time, in spite of the London fogs, which are perhaps abused to an exaggerated degree, he had never felt anything of his complaint. Yet during the two years that he spent in England, he had led a young man's life, combining work and pleasure, exposing himself carelessly to inclemencies of weather, and to the habitual causes of catarrh. Although he had caught colds during that time, he had never suffered from asthma, and his colds had even got well more quickly than when he was in France. On his return to Saint-Omer, he was immediately seized in the same way as before, and after suffering for two years, he decided on coming and consulting me. I recommended an active treatment, and sent him back to his native town. A few months afterwards, he wrote to say that he was no better. I advised him to return to Paris, but his friends answered that it was perfectly impossible he could travel as he was so ill; I still insisted on his leaving Saint-Omer. He followed my advice, and was carried to the station; before he reached Paris, his oppression had already diminished to a marked degree, and a few days afterwards, as had happened on his first visit to the capital, he was again perfectly well. I was now sufficiently enlightened as to the course to pursue, and it was useless to make a third experiment. I therefore advised him to leave Saint-Omer, and to take up his residence in London. I must add, however, that he did not follow my advice, and that, when I saw him again in 1863, he told me that he had continued to live in his native place, and that he had been perfectly well for the last five years.

A barrister, an old friend of mine, usually spends three or four months every year on his estates in Calvados. He enjoys excellent health in Paris, but no sooner goes to his estates than he has there fits of asthma, coming on usually about ten or eleven o'clock at night. His dyspnœa is so great that he is obliged to stay at his window until morning, in spite of the cold which begins to be felt during the autumn months. He is free from it in the morning, and can during the day attend to business.

I had under my care two brothers, twins, so exactly like one another that I could not tell one from the other unless I saw

them side by side. This physical likeness went further, for they had, if I may be allowed to say so, a still more remarkable pathological likeness. Thus I was attending one of them at the Néothermes, in Paris, on account of an attack of rheumatic ophthalmia, and he said to me: "At this time, my brother must be suffering from an attack of ophthalmia like myself." As I had expressed doubt at this, he showed me a few days afterwards a letter which he had just received from his brother, who was then in Vienna, and in which the latter wrote: "I am suffering from ophthalmia, you must likewise." However singular this may appear, it is a fact, which has not been related to me, but which I have seen; and I have met with other analogous instances in practice. Now, these twin brothers were both asthmatic, and that to a fearful degree. They were born at Marseilles, but they could never stay in that town, where business often called them, without being seized with asthma; they never suffered from it in Paris. Furthermore, they had merely to go over from Marseilles to Toulon, to get rid of their asthma. As they were constantly travelling from one country to another, on business, they had noticed that certain localities were fatal to them, while in others they were free from all difficulty of breathing.

This, gentlemen, is a pretty general law, and it was necessary that you should know these facts. They will be of great use to me when I come to speak of the nature of asthma.

Dr. G. Vidal communicated to me a case which fell under his own observation, and may as well be mentioned here. He knows a merchant sea-captain who has been a sufferer from asthma for many years. Whenever he goes to Peru for guano, his fits become less violent, and cease entirely as soon as he gets to the Chincha Islands; but his complaint, from which he does not suffer at all, and which he might consider radically cured, during the voyage from America to France, returns as soon as he has left his ship, and he no longer breathes an atmosphere charged with guano exhalations. This case is only in a certain measure analogous to the preceding, because it is less explicable by a change of climate than by another influence. For you know what guano is, and what a penetrating ammoniacal smell it gives off. When I come to speak of treatment, I will tell you what part ammonia sometimes plays in calming paroxysms of asthma.

There is known in England, under the name of *hay-fever*, an affection which is to a certain extent a variety of the disease of which I am now speaking. About the end of May, and during the month of June, and even at a more advanced period of the summer, some individuals are suddenly attacked with a coryza, accompanied with violent sneezing, and then with

cough and oppression at the chest, especially during the night. This curious disorder lasts sometimes with a distressing obstinacy, until the patient changes his residence, when it ceases as rapidly as it came on. I confess that I have seen several cases of hay-fever, and that I have never been able to distinguish it from asthma with periodic recurrences, these coming on much more frequently in summer than in winter. I question how far emanations from freshly cut hay has any share in the production of the symptoms of hay-fever, and whether the influence of the season is not a much more potent one.[1]

For asthma is a *summer complaint*, in this sense that sufferers from it are much more frequently subject to it in the warmer portion of the year, from May to November, than in the colder, from November to May.

Asthma, again, is more common in equatorial regions than in temperate zones or in cold climates. This fact is all the more remarkable that everybody is aware how relatively rare thoracic complaints are in hot climates where diseases of the liver and of the digestive organs predominate.

Let us enquire into what passes before our eyes with regard to the influence of temperature.

Whilst an individual liable to catarrhs dreads cold, which easily brings on his attacks, and takes good care to clothe himself warmly; whilst in winter he goes rarely out of doors, and remains by the fireside, an asthmatic person, on the contrary, likes the open air, detests small rooms, and finds that low ceilings seem to press on his chest, as it were. However wealthy he may be, you will find him generally in a room without curtains, or with curtains of light material only; heavy wool or silk hangings give him a sensation of choking and of oppression; he must often have the windows open in the depth of winter as if it were summer; in a word, he wants a considerable quantity of air. Whether this want be real, or whether it be the patient's fancy, a kind of mania, if you prefer, you will notice it, and I wished to point it out to you.

Now that I have briefly spoken of some of the circumstances under the influence of which fits of asthma arise, I will review the opinions which have passed and still pass current in the profession on the nature of this singular complaint. I will speak of the theories propounded by my esteemed colleagues, Drs. Rostan, Louis, and Beau. I will try to discuss them, and will tell you at the same time what my own views are, and how I interpret the facts.

Professor Rostan admits now the existence of idiopathic

[1] [A fatal objection to that view is the well-known fact that hay-asthma immediately disappears on removal to the sea-side.—Ed.]

nervous asthma, but he did not always do so. There was a time when he did not believe in this curious neurosis of the respiratory organs, and when he regarded it as being always a symptom of some *disease of the heart or great vessels.* Influenced by the remarkable investigations which he had made on asthma in the aged, when he was physician to the Salpêtrière, he made no difference between asthma and dyspnœa. He regarded those words as synonymous, but I am far from doing so. Asthma is, in my opinion, a special and complete disorder, a manifestation, a peculiar form of a general complaint, having very variable local expressions, sometimes giving rise to paroxysms of dyspnœa, of oppression at the chest, to a curious kind of coryza, and to peculiar catarrhal attacks, which, as I took care to tell you, may constitute the whole paroxysm; but at other times, also, manifesting itself by attacks of articular or of wandering gout, by fits of the gravel, by rheumatism, or by hæmorrhoidal affections. Asthma does not consist in oppression at the chest, because we should have to give that name not only to the dyspnœa, which is a symptom of diseases of the heart and of the great vessels, but also to the difficulty of breathing, which is so great, and which increases to suffocation in cases of œdema of the glottis, of croup, of tubercular disease, or of albuminuria. This confusion is avoided by all: there is an immense difference between dyspnœa and asthma. Although asthma is a dyspnœa of special form and nature, every paroxysm of dyspnœa is not asthma. Have you ever seen paroxysms of dyspnœa occurring in an individual suffering from disease of the heart diminish on the patient taking exercise? Do you not see the reverse every day? You may, at will, as it were, bring on a fit of asthma, or, to speak more correctly, a paroxysm of dyspnœa, in an individual suffering from an affection of the heart which is, in the least, serious. A brisker walk than usual, going up a staircase are sufficient to bring on more or less difficulty of breathing, which is in some cases so great as to give rise to a sensation of choking. I must add, however, that these attacks of symptomatic asthma may also come on independently of such exciting causes as the above; in some cases, they occur under the influence of a somewhat keen mental emotion, and in others, they appear apart from all appreciable causes.

Recall to mind the case of that woman who died a few days ago in one of my wards of hypertrophy of the heart complicating an aneurism of the arch of the aorta. You saw her on several occasions in fearful paroxysms of dyspnœa which had come on suddenly, and which towards the last recurred frequently night and day, and without any exciting cause.

If symptomatic dyspnœa may show itself, like idiopathic asthma, independently of all appreciable causes, it is important,

in order to distinguish them one from the other, that their course should be investigated.

A fit of asthma runs a course analogous to a paroxysm of fever, that is to say, it begins with a certain degree of slowness, although in some cases its access is somewhat sudden; it increases by degrees to its maximum, like all neuroses, and then decreases in the same way, until it ceases ultimately, leaving the person in perfect health, for a more or less prolonged period, until a fresh attack supervenes. This is surely not the course and aspect of a dyspnœa which is dependent upon disease of the heart. The access of this latter is generally sudden, and it never ceases so completely and so thoroughly as the feeling of oppression of asthma. It is always imminent, and does not leave the individual after a fit in a condition of perfect health, like that of the asthmatic subject, who, when his fit is once over, is not exposed to a return of it in consequence of the slightest emotion, or of a little more active exercise than usual. Until a fresh attack, which often supervenes without his being able to account for it, interferes with him, he will resume his usual mode of life and occupation, and be as free in his movements as though he were not ill. Should he, however, have secondary pulmonary emphysema, he will suffer from habitual oppression, which greatly differs from fits of asthma. An individual labouring under heart-disease will always be exposed to the risk of a fresh attack under the influence of the slightest causes. There is no doubt, and you should be aware of the fact, that paroxysms of true asthma may complicate diseases of the heart and lungs.

Let us enquire into what happens in such cases, and allow me to enter into more general considerations, to which I shall have occasion to revert more than once.

A woman, say, has cancer of the uterus, and complains of pain in the loins, and in the hypogastrium, which increase at the menstrual periods, during digestion, or when she goes to stool. Another woman similarly affected has no pain at all, while a third has uterine or sciatic neuralgia recurring daily, exactly at the same hour, with such periodic regularity that she can predict their return within a few minutes. In the case of two ladies whom I attended with Récamier and with my friend, Dr. Lasègue, respectively, these paroxysms of pain lasted five or six hours. In one of them, they continued for several years, and were of an excruciating character. When in pain, the patient rolled about on the floor. In the interval between the attacks, she had only a sensation of heat in the affected side. In these various instances, whether the pain was permanent or not, or was intermittent, the lesion was always the same; when it was intermittent, a neuralgic disorder was superadded to it; the

cancer became complicated with the painful neurosis, the existence of which it does not exclude.

In the same manner, the existence of a disease of the heart does not exclude the possibility of asthma. Some persons may be affected with most serious complaints of the central organ of circulation, without suffering from proportionately grave symptoms, whilst others, with lesions that are much less marked than those of the former, are distressed by fearful symptoms. In other cases, again, a neurosis may be superadded to an organic affection. In a word, each person, if I may be allowed the expression, has his own way of carrying his complaint. The system of this one will seem to be indifferent to the lesion, the irritability of the nervous system of that other will show itself by phenomena recurring in paroxysms, and of a peculiar character according to the nature of each individual. These are facts which one must be aware of, and you may conceive how important it is in practice to know how to distinguish a nervous from the organic element which it complicates.

What I have said of asthma and neuralgia accompanying uterine affections is also applicable to other pathological conditions.

The patient of whom I spoke just now, and who had an aneurism of the aorta, complicated with hypertrophy of the heart, had exhibited the most characteristic symptoms of angina pectoris. Now, what is *angina pectoris*? In a great many, in most cases, in fact, it is a neuralgia dependent upon an affection of the heart and great vessels, as in this woman; but it sometimes is a neurosis perfectly independent of all organic lesion of the central organ of circulation, and even of all appreciable organic lesion. It is a true epileptiform neuralgia, it is a something analogous to epileptic vertigo, and is a mode of this dreadful form of epilepsy of which I have already treated at great length. Like epilepsy, it comes on suddenly, runs a rapid course, and ceases suddenly, and it is not very uncommon for persons who have in former years had attacks of angina pectoris to become subsequently subject to epileptic seizures.

Neuroses may, therefore, be superadded to organic diseases, but they remain independent of them, and these only serve to determine their development. They are not dependent upon them, since the organic lesion does not generally accompany them, and cannot consequently be regarded as the essential condition in the production of the nervous elements of which we speak. In the case of asthma, when it comes on in individuals suffering from diseases of the heart or lungs, it is from the organic lesion determining its manifestation. I would not yet leave on your minds an idea which I do not hold, but which I

might seem to have, from what I have just stated. In asthma, the lesion may not be such as to be appreciable to an anatomist; but there is not the less a modification in the condition of the tissues, whether this modification be seated in the cerebro-spinal axis, or primarily in the respiratory apparatus, which does not perhaps alter its structure any more than an overcharge of electricity alters the glass or metallic layer of a Leyden jar.

Dr. Rostan asserts also that asthma may be due to *pulmonary emphysema*, and Dr. Louis holds the same opinion. This view is more specious than the other. For emphysema is nearly always present in asthmatic individuals, and from this the inference has been drawn that this organic lesion was the cause of the complaint, but in this as in the former instance, dyspnœa has been confounded with asthma. When cases of idiopathic nervous asthma are shown to physicians who advocate this view, they diagnose emphysema, the existence of which is often revealed by auscultation and percussion. It would be easy, however, to show them instances in which the nervous disorder does not in the least coexist with emphysema of the lungs. Thus, the patient at No. 10, in St. Agnes ward, who has been asthmatic for many years, has, it is true, both emphysema and bronchial catarrh, but this is not the case with the woman at No. 6, in St. Bernard ward. She has none of the signs of emphysema, as many of you have been able to ascertain like myself, and all over her chest, vesicular breathing may be heard of normal character.

The facts which Dr. Louis adduces in support of his views have been accurately observed, no doubt, but their importance has been exaggerated; and I will explain to you how this learned physician has been led to adopt the conclusions which he has laid down.

What are the conditions which give rise to emphysema? Is it a primary or a secondary affection? For my part, I cannot conceive it to be a primary complaint, and in order to show you how it is an effect and not a cause of asthma, I must enter into some details regarding the mechanism of its production. But, in the first place, what is the mechanism of cough? The glottis closes, after a more or less deep inspiration, and the expiratory muscles contract in order to expel from the bronchial tubes the air or mucus, the blood or pus, which they may contain. It is only after efforts which are often violent that the expiratory powers overcome the resistance opposed to them. But what takes place during this contest? Pressure is exercised from within outwards on the bronchial tubes and the pulmonary vesicles. This pressure manifests itself outwardly by turgidity of the vessels of the face and neck, towards which the blood is driven in consequence of the compression of the vascular

branches distributed through the lungs. The air which is imprisoned inside the bronchi resists the elasticity of the walls of the air-cells, and when the pressure is sustained and powerfully repeated for a long time, when the resistance from the obstacles to the free exit of the air imprisoned within the chest is too great, the walls of the air-cells get distended, the chest expands, the lung dilates, and emphysema is the consequence. In some cases even, the air-cells burst, and interlobular emphysema, into which we need not enter here, is the result.

When this mechanism of the production of vesicular emphysema is taken into consideration, no surprise need be felt at its being found in children who have had severe hooping-cough, and in individuals subject to catarrhs. Now pathological anatomy furnishes us with arguments against Dr. Louis's view, since emphysema is a very common affection, much more common than asthma, and found in the bodies of individuals who often never experienced anything during life like asthma, or, at the outside, who suffered habitually from slight dyspnœa.

Everything, therefore, goes to prove that emphysema has nothing to do with asthma. On the one hand, there is no relation between the organic lesion, which is necessarily persistent, or at least does not disappear within a few hours, and the transitory phenomena which constitute a fit of asthma; on the other hand, these phenomena show themselves without the least sign of emphysema being detectible, while again the latter may be and is, indeed, often present without the former being ever produced.

Although emphysema is not a cause of asthma, it may yet be an effect of it, and you will see how.

An asthmatic individual inspires more slowly and more deeply than the man who breathes freely, while, instead of expiring passively, as in the physiological condition, in virtue of the elastic force of the lungs alone and of the relaxation of the muscles which contracted during inspiration, he expires actively, and in a more violent manner. In spite of the efforts which accompany expiration, the air is expulsed more slowly than in the normal condition, on account of the obstacle to its passage produced by the spasmodic constriction of the bronchi which it traverses. It is conceivable then that, if asthma has existed for a long time, pulmonary emphysema may result from the efforts at expiring, recurring at each paroxysm, and being frequently also attended with cough which consists in still more energetic expiratory efforts. According to Dr. Beau,[1] asthma is the consequence of a *chronic catarrh of the*

[1] Traité clinique et expérimental d'auscultation appliquée à l'étude des maladies du poumon et du cœur. Paris, 1856, p. 156 and following.

small bronchi, accompanied by a secretion of sputa having a density and a viscous character which are only met with in this complaint. Dyspnœa arises from the presence, in the ultimate ramifications of the bronchi, of the thick mucus which prevents the exit of the air imprisoned inside the air-cells. Laennec had called attention to this kind of sputa which he calls *pearly* in the variety of catarrh which he termed *dry*. The expectoration of an asthmatic subject after a fit consists indeed of globules of mucus of the size of a hemp-seed. It is never mixed with air, is semi-transparent, of a greyish and occasionally blackish colour, while sometimes it is neither globular nor dense, and has somewhat the aspect of mother-of-pearl. Dr. Beau, who knew the view held by the illustrious discoverer of mediate auscultation, and who had personally observed cases which seemed to be in accordance with it, grounds on the existence of this peculiar expectoration of asthmatic subjects his opinion that this exceedingly plastic secretion accumulates in the bronchi. We need not, he thinks, be surprised that the patient is oppressed in his breathing, because the products of this secretion act like valves inside the bronchial tubes, just like false membranes in croup, or like foreign bodies which get into the respiratory passages. The rattling sonorous rhonchi which are then heard on ausculting the patient's chest are occasioned by the vibration of the column of air as it passes the mechanical obstacle thrown in its way by this thick mucus.

Like the previous theory which I discussed just now, this one is somewhat specious, although I think I can easily upset it.

Take a case of croup, in which false membranes have formed inside the bronchi. Although the obstacle to the free circulation of air through the lungs be then much greater than in the class of cases described by Dr. Beau, yet the paroxysms of dyspnœa from which the patient suffers have no resemblance whatever to fits of asthma. See again what takes place in the man lying at No. 10, in St. Agnes ward, who has a chronic catarrh with very profuse muco-purulent bronchial secretion.

Doubtless this muco-pus which he brings up in considerable quantities, filling several spittoons, remains in the bronchi for some time, but although the patient suffers from dyspnœa, this has none of the characters of asthma. Now it may be said that in this case the muco-pus is secreted by the large bronchi, and that there is no obstacle to the passage of air, because the calibre of the tubes is sufficiently large to allow of the free circulation of air in spite of the presence of this mucus. My answer to such an objection is that the secretion, and consequently the accumulation of mucus, takes place in the smallest bronchial tubes as well as in the large ones, and we have a proof of this in the fine mucous bubbling rhonchi which may be heard by applying

the ear to the patient's chest. The expectoration is so copious that at a given moment the bronchi in this instance are unquestionably much more completely obliterated than in the case of persons who bring up a few small pearly sputa only. Yet this man, I repeat, has nothing analogous to the paroxysms of dyspnœa which characterise asthma.

Granted that these mucous pearly sputa occasion the difficulty of breathing in a fit of asthma, it will be conceded that this secretion takes some time to form, but a paroxysm of asthma comes on with a rapidity which bears no relation to the presence of this mechanical cause. When we see a fit supervene spontaneously under the influence of mental emotion, or in consequence of the inhalation of a few grains of dust, the nature of which varies according to individuals (ipecacuanha or oats), can we assume that these various causes, which suffice to awaken the nervous susceptibility of the patient, are capable of giving rise to a mucous secretion with the same rapidity? On the other hand, you will frequently hear in asthmatic as in emphysematous subjects loud sonorous mucous rhonchi, apart from any attack of asthma, or again before or after a fit.

There are individuals also who are subject to what Laennec called *acute dry catarrh*, and who bring up pearly sputa, with extreme difficulty, after most violent efforts of coughing. They complain of a sensation of obstruction and of pricking at the aperture of the larynx and all over the chest, but which does not in the least resemble the dyspnœa of asthma.

Lastly, and this is the counterpart of what I have just stated, the catarrh which commonly accompanies asthma may be absent, and there are cases, few in number, it is true, in which no symptom of catarrh is to be seen, and no physical sign to be detected, whether the patient be examined and ausculted in the beginning of, during, or at the close of an attack.

As regards the etiology of asthma, therefore, the cartarrhal theory is as inadmissible as that which looks upon the disturbance of the respiration as an exclusive symptom of some disease of the heart or of the great vessels, and as the theory of emphysema. In a therapeutic point of view, these various opinions are not more acceptable. When I come to speak of treatment, I will tell you how inhalation of the fumes of stramonium or of burning nitre-paper sometimes suffices to stop the symptoms at once. Now would this be the case, I ask, if it were true that these phenomena were exclusively dependent upon material lesions and mechanical causes?

According to Dr. Duclos, nearly all asthmatic subjects present the herpetic diathesis. I have myself ascertained this to be a fact in a good many instances, but not in the proportion stated by Dr. Duclos. Now when asthma assumes for a few

days the continuous form to which I have called your attention, and which is accompanied by an exaggerated bronchial secretion, Dr. Duclos believes that an eczematous eruption like the one which we so often see on other mucous membranes or on the skin takes place on the pulmonary mucous membrane. That theory explains to a certain extent the strange course of this form of asthma, but it does not more than the others account for the intermittent or remittent character of the dyspnœa, which is there to testify to the existence of a nervous element.

But what is, after all, *the nature of asthma*? When one passes in review the series of facts which I have rapidly and briefly laid before you, he is tempted to compare this complaint to other spasmodic diseases of the respiratory system. Hooping-cough, of which I shall speak in my next lecture, suggests itself from the very first as an analogous disorder. An individual, for instance, gets a bronchial catarrh which, for the space of seven or eight days, has apparently the same characters as the simplest catarrh; then convulsive paroxysms come on which nothing can stop, and which recur every two hours, every hour, and even more frequently; they scarcely last from a minute to a minute and a half. In the intervals between them, the patient has exactly the same sensations as in the simplest cold; his expectoration presents nothing peculiar. The case, therefore, is one of catarrh, but of catarrh to which is superadded a nervous element which will enable you at once to diagnose the form of disease. The catarrhal element is there; occasionally it is alone present, while in rare instances the spasmodic element shows itself exclusively throughout the attack.

The same thing occurs in asthma. Although it is more commonly accompanied by all the symptoms of catarrh, and occasionally of severe catarrh, these are yet absent in a certain number of cases.

It may be justly admitted therefore, with Willis and Cullen, that asthma is a nervous affection; that the paroxysms of dyspnœa which characterise it are probably the result of a spasmodic constriction of the bronchi, which, by narrowing for a time the calibre of these tubes, prevents the free circulation of air through the lungs, and gives rise to all the phenomena which follow.

The researches of Reisseisen, which have been confirmed by more recent investigations, and in particular by those of Gratiolet, who had the opportunity of studying the anatomy of the lungs in the elephant that died at the Ménagerie, have demonstrated the existence of muscular fibres in bronchial tubes of a smaller diameter than those in which there are no

cartilaginous rings. Why should it be denied that these muscular tubes may be the seat of spasms when it is admitted that spasms may occur in other organs having a similar structure? Why should bronchial spasms be denied when no one questions the existence of vesical, intestinal, gastric, or urethral spasms?

If physiology leads one to infer their existence *à priori*, they cannot be denied when pathological cases are studied. The patient has a sensation of constriction within the chest. The energetic action of his inspiratory mucles cannot accomplish the act of breathing. It seems as if there were, and there is evidently indeed, an obstacle to the entrance of air into the bronchi; because, if you apply your ear to the chest of an asthmatic subject during a fit, you will neither hear the sound of pulmonary expansion nor the bronchial respiratory murmur which becomes audible after the paroxysm is over. And yet the muscles contract violently enough to create a vacuum inside the chest into which the air does not penetrate; the obstacle to the entrance of air is therefore in the bronchial tubes, not at the larynx, since the air passes through the glottis and traverses the trachea freely. Now, this obstacle which exists in the ramifications of the bronchi is not due to morbid secretions, as I have attempted to show, and must therefore be dependent upon spasmodic contraction of the bronchi themselves.

While admitting the nervous nature of asthma, some physicians have suggested another explanation of the dyspnœa than that of spasm. Thus, Bretonneau thought that the difficulty of breathing was due to violent congestion of the lungs. According to him, something analogous to what takes place in an epileptic seizure, attended with congestion, occurs in asthma. For if in some cases an epileptic aura is merely attended with pain, and consists in an unpleasant sensation which starts from some spot, the thumb, for example, and, ascending rapidly towards the head, is more or less immediately followed by the convulsive attack, in others the aura is accompanied by a congestive process which we can often detect. When the aura begins in the hand, this part swells, and the fingers are tightly squeezed by the rings on them. This lasts for one, two, or three minutes, and then an epileptic fit comes on. This congestion is as essentially nervous in its nature as that which causes flushing of the face during a mental emotion. Bretonneau admitted that a similar congestion occurred in asthma, and that the flow of liquids in the lungs obliterating the pulmonary vesicles and the bronchial ramifications gives rise to dyspnœa, and as a consequence to the mucous secretion which is commonly observed at the close of the paroxysms. You are aware that Cullen also ascribed to dilatation of the pulmonary

vessels the production of asthma, but, contrary to what Bretonneau taught, Cullen believed that this vascular dilatation was the cause and not an effect of the spasm.

In spite of all my respect for the views of Bretonneau, who was my first and excellent teacher, I have always opposed this theory. I have never been able to understand the aura epileptica, but I do understand the mechanism of mere spasmodic constriction, and, more than this, I cannot conceive how it could be different.

Thus, gentlemen, asthma is a nervous complaint, a neurosis; and, in order better to define the class to which it belongs, I will add, a diathetic neurosis; that is to say, it very rarely happens that it does not depend upon the existence of a diathesis. I will endeavour to prove this by facts, and allow me, therefore, to enter into some details which will not be devoid of interest.

An individual, say, becomes asthmatic when about thirty or forty years old. Until then, he had been entirely free from that complaint, and vain attempts are made to discover an exciting cause; but on enquiring into his previous history it is made out that the patient exhibited in his youth symptoms of a different kind which were evidently manifestations of a diathesis. These were eruptions of an herpetic or, more commonly, of an eczematous nature; or he had rheumatic pains; or again, at a more advanced age, he had either fits of the gout or hæmorrhoids. Indeed, nothing is more common than to find herpetic, rheumatic, gouty, or hæmorrhoidal affections transform themselves into asthma. The fact was pointed out long ago. Truka,[1] and Musgrave[2] have both recorded instances of the kind; and in reference to gout, I have known a case in which attacks of arthritis alternated with fits of asthma with great regularity: fits of the gout sometimes followed one another; at other times, paroxysms of asthma; occasionally, an attack of gout came on after one of asthma, or reciprocally, but the patient never had gout and asthma simultaneously.

Thus eczematous eruptions, rheumatism, gout, and hæmorrhoids, and I may add, gravel, are complaints which may be replaced by asthma, and may replace it in turn: they are different expressions of one and the same diathesis. Hemicrania is another affection which I must not omit.

Many individuals who are subject to periodic headaches are, or have been, affected with gout, rheumatism, or eczematous eruptions, or are the issue of parents who were so affected; or you may notice the reverse; that is to say, you may see eczema-

[1] Historia hæmorrhoidum. Windobonæ, 1794.
[2] Traité de la goutte.

tous and hæmorrhoidal affections, attacks of rheumatism or of gout, following on periodic headaches. Among other examples of these diathetic transformations, I will relate to you the following, which was the first case of the kind which attracted my attention at the commencement of my medical career.

I was intimately acquainted with a Major of the English Army who for a long time had been subject to headaches that recurred so periodically—every other Wednesday—that he knew within an hour when he was going to be attacked. His seizures were so regular as to their course and duration that, more extraordinarily still, he could tell when they would terminate. They indeed lasted a few hours, and then left him in a condition of perfect health. He had become subject to these attacks while staying in the West Indies; since then, they had never missed until the time when I made his acquaintance in Paris. He was quite tired of his complaint, and begged me one day to rid him of it. This was in the year 1824. I did not know then what hemicrania was. I asked the advice of some other practitioners, and prescribed blue pills in large doses. Under the influence of repeated purgation, the attacks ceased to be periodical, but his general health was far from improving. Formerly, at the termination of the attacks, he felt perfectly well, and his sensations contrasted singularly with the *malaise* which he felt when the attacks were on the point of coming. In fact, he felt like all those who suffer from a gouty or hæmorrhoidal diathesis, and who feel such relief from the attacks, which are often preceded by an undefined *malaise*, that the seizures seem to be indeed necessary evils.

My patient went to reside at Fontainebleau during the fine weather, and I used to go and spend a few days with him there from time to time. One morning, he had me called up in order to show me his foot in which he had excruciating pain. I noticed swelling and considerable redness of the foot; in fact, he had a fit of well-marked acute gout. I did not at that time know how much one should respect such manifestations, and I was not aware that gout and headache are sister diseases: influenced also (in spite of myself and in spite of the principles which I had been taught when I began to study medicine) by the doctrines of Broussais, which were then in full vogue, I decided on adopting an antiphlogistic treatment. I ordered leeches, and emollient poultices sprinkled with laudanum to be applied to the painful part. The arthritis yielded, but from that day, the patient lost his former good health. He had a second attack, but of indolent, atonic gout, and not only did his general health fail, but even his mental and intellectual condition became deplorably affected. He lost his usual vivacious and merry ways, became dull, cross, and disagreeable; at last, he had an

apoplectic stroke, and died two years afterwards of a second attack.

I might relate a great many similar cases, but will confine myself to three more which bear more especially on this point.

On July 15, 1861, a young man aged thirty, and residing habitually in the department of the Côtes-du-Nord, came to consult me in Paris. Dr. Blondeau, who happened to be in my consulting-room at the time saw him with me. He had the appearance of a man enjoying perfect health, and stated that no member of his family had suffered from gout. When a boy, between ten and fifteen years old, he had a moist eruption on his legs which disappeared somewhat suddenly to show itself again ten years later. But from the age of seventeen to twenty-one, he had been subject to frequent paroxysms of nervous asthma which were so violent as to place him at death's door. He was only relieved by bleeding.

At the age of twenty-one, he had fits of regular gout, and from that time his asthma left him. However, as he was impatient of pain, and was anxious to get rid of it at any price, he had recourse to preparations of colchicum, and those fatal secret remedies, the Sirup of Boubée, Lartigue's pills, and Laville's Liquor, which are efficacious, but dangerous also. He got rid of his gout, but in less than three years, his health had become seriously impaired, and he looked prematurely old. He then went to Tours and consulted Bretonneau, who at once made him stop the dangerous remedies which he had been taking and advised him to take the Carib ratafia (made of taffia and guaiacum root) and to take a good deal of exercise and live well. Under the influence of this plan of treatment, his acute gout returned and his health with it. Sometime afterwards the patient went to Bagnères-de-Luchon, where he drank the waters and remained free from gout for two years and a half. The fits then returned, but were less violent; and when I saw him, he had had none for eighteen months. He complained however of headache, which returned every ten days or fortnight; always beginning in the right temple and stopping at the occipital region on the same side, and lasting about three or four hours. His health was excellent, with the exception of these headaches which, if I may be allowed the expression, were the small change, as it were, of the fits of regular gout.

On March 2, 1863, I saw in my consulting-room again a man aged thirty-five, who had been subject to asthma since the age of sixteen. His attacks were never less than a fortnight's duration; they came on especially when he stayed in Paris and were very rare if he lived in the country. For the last six months they had grown less severe, and the last attack which

had set in with very moderate violence, had terminated on the third or fourth day in a fit of perfectly regular gout.

In the following case, which was communicated to me by my colleague Dr. Hérard, it was not gout but rheumatism which was followed by asthma.

A lady about fifty years old had been attacked for the first time when about thirty years of age, by acute articular rheumatism a few weeks after confinement. She recovered, but had a relapse at the end of two months. From that time she became subject for several years to vague, wandering, rheumatic pains in the muscles.

These pains ceased. After this, other neuralgic pains showed themselves, and the patient became at the same time subject to periodic headaches from which she had never suffered previously. She came under Dr. Hérard's care in 1858. She was then troubled with a spasmodic cough, which recurred regularly every night at the same hour. In the course of that winter, she had facial neuralgia, and the skin of her neck became covered with a dartrous eruption, of a papulo-vesicular character, which lasted a short time only.

She went through the next winter without illness, but in 1860 she had fits of well-marked nervous asthma, which came on in the evening and during the night. Dr. Hérard noted a peculiar circumstance, which has great analogies with what I told you of the influence of climates, namely, that the paroxysms of asthma came on especially when this lady stayed in a certain quarter of Paris. She lived in Cirque Street, and she had generally an attack when she went to spend an evening in the neighbourhood at her brother's, in Ville l'Evêque Street. These paroxysms, which remained during fifteen or twenty days, never returned again, and were never accompanied by symptoms of catarrh.

These facts might, perhaps, be explained in the following manner. When persons suffering from gout or from piles do not in the proper season exhibit the usual manifestations of their diathesis (fits of articular gout and bleeding piles), they become, in a great many cases and to a very high degree, subject to nervous symptoms, such as spasms of the stomach or the intestines, a condition of general *malaise*, which expresses itself by moroseness, sadness, or some change or other in their temper. These phenomena, it is true, often also precede, although to a less degree, the regular attacks. Now, it may be questioned whether asthma is something else than a form of these spasmodic affections which then attack the lungs,

This is the theory propounded by Dr. Duclos; and the following case, which resembles the preceding ones, supports it, in a certain measure.

I had under my care, a lady about thirty years of age, whose fits of asthma coincided with an eruption of urticaria. They lasted two months, and when the urticaria disappeared, the feeling of oppression at the chest increased invariably; so that it might justly be supposed that the asthma was caused by an exanthematous eruption in the bronchi.

There is another diathesis which differs from those to which I have just called your attention, and of which asthma may also be a manifestation, namely, the tubercular diathesis. Of the numerous instances of the kind which have fallen under my observation, I select the following, one of the most remarkable of them, the case of a lady, aged seventy, who enjoys very good health, with the exception of occasional fits of asthma. Her mother died of phthisis, and she has lost two daughters, one of whom was carried off by cerebral fever, and the other died of phthisis.

Tubercular individuals may, therefore, give birth to asthmatic children, and, on the other hand, asthmatic subjects may have tubercular children. It is certainly a very remarkable fact that asthma, which seems to be such a trifling affection as regards the organic lesion which accompanies it, should apparently be, in some cases, a manifestation of a diathetic disease so marked in its localisation as tuberculosis is. Bear these cases in mind, gentlemen, for they have reference to a great question, namely, the transformation of morbid affections into one another, a vast subject which we cannot enter into here, and which might be treated in an important chapter of general pathology. In reference to the point which we have just been considering, remember that eczematous and rheumatic affections, gout, gravel, hæmorrhoids, periodic head-aches, and asthma, which are varying expressions of one and the same diathesis, may replace one another. In proportion as you make further progress in the practice of your profession, you will but too often have the opportunity of verifying the accuracy of this statement.

Lastly, like all diathetic diseases, asthma is directly transmitted from parent to offspring.

The case of a man who has been for a long time an in-patient here, is sufficiently typical to serve as an illustration of this point.

He is thirty-one years of age, and has been subject to fits of asthma since he was thirteen. He had been perfectly free from them until then. He associated with other children of his own age, joined in their games, running and going through the same exercises as the others, without feeling the least distress, when he had a first attack of asthma, without his being able to assign any cause for it. The fit came on at three o'clock in the afternoon, and lasted from four to five days. From his

description, it assumed the catarrhal form, and was so severe as to alarm his friends and the medical men who were consulted.

Five years afterwards, the symptoms became more regular; they no longer came on during the day, but always after twelve at night—about one or two o'clock in the morning. You already find here a fact on which I have laid much stress, namely, the period of day at which the paroxysms occur. The patient stated of his own accord, without being led by me, that these attacks were commonly of very great violence, if previously to his getting into bed he shook his paillasse, and he ascribed the intensity of the paroxysm to the presence of the dust which he raised about him in the room. There was generally but one paroxysm at each attack, and he was free for six months. At the present date, they return every six weeks, and last three days, that is to say, during the three days he has constantly a sensation of constriction and distress about his chest, which prevents him from working; this symptom becomes more intense at night, and generally diminishes in the early morning, although it occasionally increases then.

The patient mentioned also a circumstance to which I am desirous of calling your attention, namely, that his expectoration presents perfectly different characters before and after a paroxysm. During the fit itself, he does not expectorate at all; before it, he brings up small, thick, globular sputa, which he compares to the germ of a hen's egg. You will recognise pearly sputa by his description. After the attack, his expectoration becomes mucous and purulent; you have yourselves seen it in the spittoon, and it differed in no respect whatever from that of an individual suffering from the most genuine catarrh. This case, in which pearly sputa are brought up before a fit, where there is consequently no distress of breathing, no oppression at the chest, no asthma proper while the dyspnœa is unattended with expectoration, and the catarrhal expectoration occurs after the difficulty of breathing is over; this case, I say, is another instance which tells against Dr. Beau's theory.

This case illustrates also what I have told you of the various forms of the disease in infancy and adult age, and of diathetic transformations. The patient's mother was gouty, his father epileptic, and he was himself subject for some time to periodic headaches.

In the intervals between the paroxysms, he could go through the most violent gymnastic exercises without feeling the least distress of breathing. On several occasions he betted with his friends that he would go from Paris to the Place d'armes at Versailles (a distance of eighteen kilometres, between thirteen and fourteen miles), and keep up with the Versailles omnibus which, as you know, goes at a pretty quick pace.

He can run therefore for about an hour and three quarters, walk pretty briskly up the rather steep ascent of Sévres and of Chaville, and reach the end of his journey without feeling more out of breath than a man might be who had just walked in measured step for the distance of half a kilometre. You may remember that during his fits he had all the physical signs of vesicular emphysema of the lungs: the inspiratory murmur was almost *nil*; expiration was forced and longer than inspiration; there were sonorous rhonchi and exaggerated resonance of the chest. When he had been free from asthma for a few days, this inspiration became longer, full, and easy; the vesicular murmur perfectly normal; expiration less prolonged than inspiration, performed without effort, and the thoracic resonance less than during the attacks. (In children and in adults suffering from hooping-cough, you will likewise frequently find signs of vesicular emphysema, which will rapidly disappear as soon as the neurosis itself shall have passed.)

Lastly, the history of this patient is again complete with regard to the treatment of the asthma. When a paroxysm is coming on the patient gets out of bed, heats some water and takes a foot-bath, which generally relieves him; at other times, he is obliged to stand at an open window, however bad the weather may be, so as to allay his sense of anxiety by breathing the fresh night-air. If asthma were a catarrh, would such a plan of treatment succeed? Stramonium gave him little relief, and ammonia, of which I shall speak presently, caused him much inconvenience. He derived, on the contrary, much benefit from arsenic. In this case, you have an illustration of the therapeutic fancies of asthma, as in others you had of its pathological caprices.

Indeed, like all neuroses, asthma often yields to measures which differ widely according to individuals; and experience alone teaches the patient and the practitioner what those measures are. I have already stated that asthmatic subjects commonly sought after fresh air; others, on the contrary, can find relief in their paroxysms only by standing with their back to a roaring fire. The last patient I alluded to, derived benefit from a hot foot-bath. If I were to enumerate all the plans, some more curious than others, which certain persons have recourse to, the list would be long before it came to an end. The brother of the Chancellor of the late Chamber of Peers had from four to six Carcel lamps lighted in his apartment whenever he had a fit of asthma, and felt immediately relieved after this. Another patient, whose attacks occurred during the day, got on horse-back, and could only calm the paroxysm by galloping in the teeth of the wind.

These are surely singular and exceptional facts, but it was important that they should be mentioned, because they afford

fresh proofs of the essentially nervous nature of the complaint.

I now pass on to the subject of *treatment.*

In certain countries in which asthma is a common disorder, its treatment was formerly empirical. In the East Indies, the popular remedy consisted in smoking the leaves of the *datura metel.* Dr. Anderson, who was in practice at Madras, recommended, and gave a quantity of them to an English officer who brought them to Europe in 1802, and made a present of them to Dr. Sims, of Edinburgh. The latter, on finding that the smoking of these leaves was followed by good results, tried to substitute for them *datura stramonium.* The trials answered, and stramonium has now become a popular remedy for asthma.

What I shall say of *datura stramonium* is applicable to the other species of datura, the *datura ferox, fastuosa,* or metel, although the first is the one generally used. Of all remedies which have been tried in asthma, stramonium generally answers best. The dried leaves of the plant are smoked either alone or mixed with sage, in a pipe, or rolled up in cigarette papers, or they are burnt in the patient's room. All asthmatic subjects are not however relieved, and habitual tobacco-smokers often derive no benefit from it. This is conceivable since tobacco is a solanaceous plant like stramonium, and, a man used to tobacco may not be amenable to the stupefying influence of stramonium. I know, however, tobacco-smokers whose paroxysms are calmed by stramonium; so that this would show that the latter has a specific virtue somewhat different from that of nicotine. Although one of these drugs does not, consequently, replace the other completely, there are still individuals who are not habitual smokers, and who obtain relief by smoking tobacco; I do so for my part, and I have already told you that when I had a fit of asthma, I had only to draw a few puffs from a cigar to remove the dyspnœa. Generally speaking, *all solanaceous plants,* stramonium, tobacco, hyoscyamus, and belladonna, are therefore more or less endowed with the same properties; they are all used in the preparation of the Espic cigarettes, which have long enjoyed, in the treatment of idiopathic nervous asthma, and of pulmonary catarrh complicated with nervous symptoms, a reputation which is still trumpeted by newspaper-advertisements. They are prepared according to the following formula, which you will find in some of your text-books:

℞: Folii optimi belladonnæ	gr. vj.
„ „ hyoscyami	gr. iij.
„ „ stramonii	gr. iij.
„ „ phellandrii aquatici	gr. j.
Extracti opii	gr. ¼
Aquæ distillatæ lauro-cerasi	q. s.

The leaves, after careful drying and removal of their veins, are cut up and thoroughly mixed. The opium is dissolved in the cherry-laurel water, with which the leaves are wetted. The paper used for the cigarettes is also soaked in the solution and then dried.

You may conceive the efficacy and success of such a combination of remedies. Yet, when stramonium and other solanaceous plants are prescribed, the patient should be particularly warned against abusing them, lest he should quickly exhaust their influence. He should have recourse to them only when the attack is violent, and should then smoke two cigarettes during the fit, and not seven, eight, or ten, as many are tempted to do. When the patient cannot smoke, or does not know how, stramonium may be burnt in his room, and thus the air in it charged with the fumes of the drug.

With this plan of treatment, as with all those which are used against nervous disorders, idiosyncrasies should be taken into account. One asthmatic subject, for instance, will be relieved by stramonium, another by belladonna, a third by hyoscyamus, and a fourth by tobacco or a mixture of these various plants. In some cases, as in that of the man whose history I related to you in detail, solanaceous plants cannot be borne, so that other methods of treatment have to be had recourse to, some of which have been justly lauded, as, for example, *arsenical cigarettes*, and the fumes of burning nitre-paper. To prepare arsenical cigarettes *twenty grains of arsenite of potass* are dissolved in *half an ounce of distilled water*, and a sheet of bibulous paper is soaked in this solution until it is entirely taken up. The paper is next dried and divided into twenty equal pieces, which therefore contain about one grain of arsenite each. Each piece of paper is then rolled up into the shape of a cigarette, which the patient smokes after lighting, and, by inspiring slowly, draws in the smoke inside the bronchi: he should take five or six puffs only, once a day.

The nitre cigarettes are prepared in the same way, by soaking some blotting paper in a nearly saturated solution of nitrate of potass.

If the patient does not know how to smoke, the arsenical or nitre-paper is rolled up into a ball and ignited, and the patient inhales the fumes through a funnel-shaped tube of paper held over the ball.

I have sometimes combined nitre and stramonium, or belladonna fumes, either by rolling leaves of these plants within the paper impregnated with the solution of nitre, or by directly dipping the leaves in a solution of nitre. Of the measures directed against a fit of asthma, there is one which has been, in turn, lauded and rejected too absolutely; for when properly em-

ployed it has been of real good service; I allude to the topical application of ammonia to the posterior part of the pharynx.

Ducros, of Sixt, was the first to propose this plan of treatment. He treated all asthmatics by brushing the back of the pharynx with a mixture of equal parts of water and liquid ammonia.

Ducros was a curious individual, and held the queerest medical theories, and he was led to adopt the above plan on account of his singular idea that the back of the pharynx was the centre from which emanated the nervous power, the influence of which he tried to modify. Strange as his starting-point was, he was in some cases successful, and his success in the case of Madame Adélaide d'Orléans, sister of the king Louis Philippe, gave him for a time a very great reputation in Paris. Trials made by other medical men—by Dr. Rayer and by myself—proved the efficacy of the remedy in certain cases: but it occasionally gave rise also to formidable accidents. I must, therefore, while admitting the good effects of this plan of treatment, warn you against the risks attending it.

Two cases among others made a lasting impression on me.

A man, of colossal stature, consulted me one day on account of asthma; he had been sent to me by my friend Dr. A. Lebreton. I tried Ducros' method, but on my first attempt, just as I had passed down to the back of the fauces a brush dipped in a mixture of equal parts of water and liquid ammonia, the patient had an awful paroxysm of orthopnœa. He started up on his feet, as if pushed by springs, and rushed to the window in a truly fearful state of suffocation. He thought that he was going to die, and I confess that I thought so too. He grew quieter, however; but neither of us cared to repeat the experiment.

Some time afterwards a lady, whom I have had occasion to see since, consulted me for the same complaint. I again tried the ammonia treatment, but this time I took the greatest possible precautions. Notwithstanding these, I had scarcely touched the pharynx with the brush, than a fit of choking supervened. The result was successful, nevertheless, for the patient was free from asthma for the next two months, which was very unusual with her.

Lastly, the patient in St. Agnes ward has stated in your presence that he had been treated in that way once, but that he had been seized with such oppression at the chest that he thought he was going to die. From this time, besides, he had fits of asthma every four days, which recurred at the same hour that the topical application of ammonia had been done, whilst, previous to this, his attacks returned every three months only.

Ducros' method, therefore, serves only a few patients, although a great many bear it without any inconvenience. Ducros himself had recourse to it every day, and never, he said, with unpleasant results. The cases which I have just related show, however, that you cannot be too prudent, for one may understand that death might take place in one of these fearful paroxysms. When I have recourse to this method of treatment, I therefore take a precaution which I recommend you to adopt. I first make the patient inhale some ammonia from a bottle, and after this, I apply to the back of the throat, on the first occasion, a solution of one part of liquor ammoniæ to nine of water. The next day I use eight parts of water to one of ammonia; and I diminish the quantity of water by degrees to one-third, until the patient has grown accustomed to it, when I use equal parts of water and liquor ammoniæ.

Another method of applying this treatment consists in placing in the room where the patient is, dishes containing liquor ammoniæ, the vapours of which are diffused in the atmosphere of the room. Dr. Faure's method is in some respects different from the one which I have just described, and you saw me have recourse to it in a patient at No. 22, in St. Agnes ward.

This man was subject four years ago to fits of asthma which returned every night at the same time, and lasted about two hours. For nearly three years and a half, he had had no attacks, when in the course of the spring of the year 1860, these returned again. He was seized during the night as previously, but the dyspnœa continued during the day, and on examining his chest, I recognised pulmonary emphysema. He never brought up pearly sputa, however, and his expectoration resembled lightly boiled white of egg.

I prescribed for him ammoniacal inhalations, according to Dr. Faure's method, which consists in holding one's mouth at a distance of about a foot over a vase containing a table-spoonful of liquor ammoniæ: the inhalation to be continued for a quarter of an hour, and to be repeated four times in the twenty-four hours. The patient's nostrils had to be stopped up with some cotton wool, because he could not otherwise bear the smell of the ammonia.

You saw that from the first day the nocturnal paroxysms disappeared, the diurnal dyspnœa ceased almost entirely, and the patient seemed perfectly well after four days of this simple treatment.

It is probably to these vapours, that some asthmatic subjects owe the relief which they experience by merely staying for some time in places where there is a disengagement of ammoniacal gas. I have already mentioned the instance of that merchant sea-captain who was free from asthma so long as he

was on board his ship with a cargo of guano, or when staying at the Guano Islands.

Antispasmodics, such as ether, either given with simple syrup or in capsules, are also indicated during the fit.

In some cases, which I have already defined, I have obtained good results from the timely administration of an *emetic*, and ipecacuanha is the remedy to which I then give the preference.

Now that I have described some of the methods of treatment which are useful during a fit of asthma, it remains for me to tell you how to prevent the recurrence of the attacks. Medical interference is unfortunately very often powerless in that regard, for we can better moderate the symptoms of the complaint than cure it radically. The following, however, is a method of treatment which seemed beneficial to me. It consists of a certain number of series:

1. For ten successive days, every month, the patient takes at bed-time first one, then, after three days, two, and for the last four days, four pills like the following:—

℞: Extracti belladonnæ } gr. ½
Pulveris radicis belladonnæ } âā
fiat pilula una.

or from one to two, up to four granules of atropine, containing one-fiftieth of a grain each of the drug.

2. During the next ten days, the syrup of turpentine, in doses of a table-spoonful three times a day, or better, three capsules of spirits of turpentine are substituted for the preparations of belladonna.

3. For the last ten days of the month, the patient is made to smoke arsenical cigarettes.

Lastly, to complete the treatment, the patient takes every tenth day, in the morning fasting, a drachm of powdered yellow cinchona bark in a cup of coffee.

The essential part of the treatment consists, I believe, in the internal and prolonged administration of belladonna or atropia, according to the formulæ and to the rules which I have just laid down. It is not necessary that the patient should feel, to a marked degree, the physiological effects of these powerful drugs, but his system should for a long time be kept under their influence. Bark, turpentine, the inhalation of arsenical vapours are, however, very useful adjuncts.

This method of treatment is far from being infallible, although it has given me, and gives me every day, excellent results. It is successful in some cases, but fails entirely in others, so that other measures have to be adopted.

Dr. Duclos, of Tours, asserts that *flowers of sulphur* are a remedy "of prodigious efficacy" for the prevention of asthma.

He prescribes it "in daily doses of from ten to twenty grains, according to the age of the patient, to be taken in one dose in the morning early or just before breakfast. This dose is repeated for five or six months, during twenty days every month, then for a year, eighteen months or two years, for ten days every month." This treatment is, he considers, the most simple and the most easy to carry out that can be imagined. The disease was improved in every case in which he had recourse to it, and he cured a great many individuals.[1]

Dr. Duclos insists on the necessity of giving flowers of sulphur instead of any other preparation of sulphur. It is not an indifferent matter, in his opinion, as to the choice made; for while he has obtained good results from the flowers of sulphur, he had constantly failed when he used to prescribe sulphuretted waters, like those of Barèges, of Bonnes, and of Cauterets. He remarks, *à propos* of this, and justly too, that among the substances used in materia medica, there are analogues and not succedanea; that sulphuretted waters are not flowers of sulphur, no more than cinchona bark is the sulphate of quinine, or opium, morphia.

I have myself treated asthma successfully with sulphur; but I cannot share the illusions of my learned colleague of Tours with regard to the infallibility of this remedy in all given cases. Sulphur seems to me to be clearly indicated, and to be of unquestionable service, when the asthma is due to an herpetic diathesis; but if we exaggerate the importance of facts, and draw general conclusions from particular cases, and fancy that the treatment should be always exactly the same in every case, we run the risk of being sorely disappointed.

In those cases in which sulphur is of real benefit, namely, when the neurosis of the respiratory apparatus is dependent on an herpetic diathesis, *arsenic given internally* has been in my hands, and is still, of very great service. No surprise need be felt at this, if the marvellous effects of arsenical preparations in the treatment of herpetic disorders in general, and skin diseases in particular, be taken into account.

Arsenic is not only very efficacious in such cases, but it is also an excellent remedy against asthma in a good many other instances which have nothing to do with the herpetic dia-

The remedy is not a new one besides. I need only mention that Dioscorides administered it in asthma, either mixed with honey or in a mixture containing resin. He used what was in his day called *sandarach*, but is now known as realgar or red

[1] Recherches nouvelles sur la nature et le traitment de l'asthme (Bulletin général de thérapeutique, Avril 15, 1861, t. lx., p. 299.

sulphuret of arsenic. At a less remote period from us, at the end of the sixteenth century, George Weitth lauded an electuary which contained orpiment (the yellow sulphuret of arsenic), of which he gave a large dose every day to patients suffering from the gravest forms of asthma. The use of arsenic, however, was obstinately opposed by the majority of medical men; the mineral was absolutely rejected from materia medica, and it was thoroughly discredited, when, in our own time, Harles attempted to bring it into favour again. It enjoys now full favour, and deservedly holds an important rank as a therapeutic agent.

In reference to its use in asthma, you have doubtless heard of the arsenic-eaters, or toxicophagi, of various parts of Germany, Lower Austria and Styria. In those countries the peasants, and even the townspeople, are in the habit of taking several times a week, in the morning fasting, at first a small quantity—about half a grain—of arsenic and by degrees larger doses. By so doing, they hope to make their complexion look fresh, and to grow moderately stout and also, to become, to use their own expression, more *volatile*, that is to say, to increase their breathing capabilities when walking up hill. When they have to walk a long distance in the mountains, they hold in their mouth a piece of arsenic of about the size of a small lentil, and let it melt by degrees. The results of this practice are really surprising; they can by this means ascend with facility heights which otherwise cause them great fatigue. The men not only eat arsenic themselves, but also habitually mix it with the food of their horses, especially those which work their carts in mountainous districts.

Profiting by the example of these people, some medical men asked themselves whether this singular and quite special influence of arsenic on the function of respiration, might not be utilised in the treatment of certain respiratory disorders. Dr. Koepl was one of the first who, accordingly, thought of trying the liquor Fowlerii, or arsenicalis, in cases of asthma, and he obtained the good results which he expected in a pretty good number of cases. Others tried the same remedy, and with similar success; and for several years I have myself given arsenic internally in spasmodic asthma with unquestionable success.

I commonly prescribe the *arseniate of soda* in a mixture like the following:

℞:	Sodæ arseniatis	gr. j.
	Aquæ destillatæ	ʒiij.
	Tincturæ cocci	q. s. (ad colorandum).

The patient is made to take every day, at the commencement

of his two principal meals (a useful precaution in order that the remedy be well borne by some irritable stomachs) a *tea-spoonful* of the solution, containing the twenty-fourth-part of a grain of arseniate of soda.

I also prescribe *arsenious acid* in pills:

℞: Acidi arseniosi gr. v.
Amyli gr. c.
Syrupi acaciæ q. s.
pro pil. centum.

Each pill will contain one-twentieth of a grain of arsenious acid, and is to be taken before each of the two principal meals in the day. When I have to deal with timid individuals, who may dread the idea of taking arsenic internally, I call these *Dioscoride's pills.*

According as it is tolerated by the patient, I increase or diminish the dose of the medicine, and I continue it for several months in succession, generally intermitting it for eight or ten days every month.

There is another treatment for the cure of asthma, about which I should say a few words. You have seen me for some time give *iodide of potassium* to two men in St. Agnes ward who presented perfect types of spasmodic asthma. The history of this method of treatment is rather curious.

Two or three years ago, a French medical paper published an extract from a foreign journal[1] in which it was stated that a secret remedy for asthma, of which iodide of potassium was the principal constituent, was being sold at Boston.

The author of the statement, Dr. Horace Green, added that he used the remedy with the greatest success, especially in cases of asthma complicated with bronchitis, and gave the following formula:

℞: Potassii iodidi gr. xl.
Decocti polygalæ ʒiij.
Tinct. lobeliæ } ʒ iv.
„ Camphoræ cum opio } āā

Two or three table-spoonfuls a day.—

About the same time, Dr. Aubrée, now practising as a pharmaceutist at Burie (Charente-Inférieure), wrote a letter to the Academy claiming the priority of the discovery, and addressed to me another letter, giving me the credit of the method of treatment which he had used for the last fifteen years, or at least part of the credit. I have preserved his letter, of which the following is an extract:

[1] Favourite formulæ of American practitioners, by Horace Green, of New York, translated into French by M. Noirot (1860) (Schmidt's Jahrbücher der gesammten medicin), Band 114, No. 112.

'About fifteen years ago, when I was residing in a small town in the Hérault department (Pézenas) I was consulted by a man from the neighbouring village of Velleros on account of a well-marked neurosis of the respiratory organs. He showed me a prescription by you for tincture of iodine which was to be rubbed into the axillæ, the pit of the stomach, and the back, and for a mixture of iodide of potassium (one hundred grains to eight ounces of distilled water, sweetened with simple syrup). I repeated the same treatment, and he felt better from the second day. He came back to me rejoicing after a few days, but he was not perfectly well yet. I told him to cease the frictions, and doubled the quantity of iodide of potassium in the mixture. The wheezing in his chest disappeared completely, his breathing recovered its usual rhythm, and from that time he never had another attack of the same complaint.'

Encouraged by this case, Dr. Aubrée tried the same remedy in a great many other instances, and gave an *anti-asthmatic elixir* prepared according to the following formula:

℞ Poligala root	gr. xl
Boil in	
Water	℥iv.
Reduce by boiling to	℥ij.
Filter and add:	
Iodide of potassium	ʒiv.
Syrup of opium	℥iv.
Brandy	℥ij.
Colour with	
Tincture of cochineal	q. s.
Filter.	

The patient should take three table spoonfuls of this elixir in the morning fasting, at noon, and in the evening until the asthma disappears. As some individuals do not bear the remedy well, (and this is not surprising because each dose contains about forty-five grains of iodide of potassium and four-fifths of a grain of extract of opium.) Dr. Aubrée makes it an indispensable condition to suck after each tablespoonful a chocolate pastille, which neutralizes the irritating action of the iodide of potassium. He states that he has by this means cured in the short space of three days, twelve persons.

Since this communication from Dr. Aubrée, and since the secret remedy of certain quack medic nes has been made known, I have very frequently given iodide of potassium, but have modified the formula as follows:

The patient takes every day immediately before dinner a teaspoonful of a solution of

Iodide of potassium	℥iij.
Distilled water	℥vj.

I must say that I have obtained with this remedy successful results, in a great many cases where other methods of treatment had failed. On the other hand, we must not be wilfully blind, for I have known the iodide not only fail but also increase the disease markedly, as it did in the case of the two men in St. Agnes ward.

One of them was, on the contrary, immediately relieved by *chloroform inhalations*. He was driven to try them through the obstinacy of his fits; the relief he experienced induced him to try it again, and he had come to abuse it so as to consume in one day sixteen ounces. He thus spent all his money, while his health became also deeply impaired. His liver got out of order; he had several attacks of severe jaundice while he was under my care, although he only used then a comparatively small quantity of chloroform, about five ounces at the most, in the twenty four hours. At the time when he was using such large quantities of the drug, he had fallen into a state of acute mania like *delirium tremens*, and had been compelled to interrupt such dangerous inhalations. They had a marvellously rapid influence on the fits of asthma, calming them completely in less than a minute, although they returned in a short time, so that chloroform had to be administered again.

In some cases, when the paroxysms were much less violent than in this man, I have sometimes seen slight inhalations of chloroform suffice to calm the fit entirely, in the same way as in other instances a few puffs from a stramonium cigarette produce rapid and complete results.

I have dwelt so much on the treatment of asthma, gentlemen, because one plan of treatment cannot be made to include all cases. Strange differences exist on this point; and whilst one man gets well almost at once, another who is apparently in a similar condition, derives no benefit and even feels worse from the use of the same remedy.

There is no inconvenience, however, and it is often advantageous, to combine these various remedies as I do now. And I will give you an instance of my practice.

On December 2, 1862, I was consulted by a young married lady, twenty seven years of age, who had been subject to asthma since she was seven or eight years old, and whose fits were so frequent as to scarcely leave her free for a fortnight in the space of three months. I prescribed arseniate of soda at breakfast-time, iodide of potassium at dinner-time, belladonna in the evening, and every week, in the morning fasting, a dose of two drachms of pulvis cinchonæ flavæ. On July 1, 1863, this lady called on me again, and informed me that she had not had a single attack for a long time.

Let me say a few words more before I conclude as to

the choice of localities about which you will certainly be consulted.

When I spoke of the exciting causes of asthma, I told you of the influence of climates and localities on various individuals, and I related cases of persons who had fits of asthma only in certain countries, while others were continually subject to the disease. You should turn to advantage the knowledge of this fact; but when you counsel your patient to change his residence, you should appeal to his personal experience, or warn him, if he has not previously tried this plan which is often so efficacious, that he must be solely guided by his experience. There is indeed no absolute rule on this point, as one locality which will agree with one will not with another. Thus, low places generally agree with persons whose respiration is hard, as it is termed, while high grounds disagree with them. Yet I knew a superior officer who had frequent fits of asthma when he resided in Paris, but was free from them for ten months that he spent at Clermont-Ferrand, and had not the slightest oppression at the chest all the time that he stayed in the mountains of Mont Doré, where he made many excursions on foot and on horseback.

It seems that the elevation of the district where the patient resides exerts an influence which one would hardly have suspected. A resident assistant of the Beaujon Hospital, which is situated in the Faubourg Saint-Honoré, had constant attacks of asthma. He obtained Professor Marjolin's consent to exchange with one of his friends who was at the Hôtel-Dieu, a hospital built, as everybody knows, on the banks of the Seine, and in the lowest part of Paris. He never had a fit at the Hôtel-Dieu, but whenever he went to dinner with his old colleagues at Beaujon, he became at once oppressed in his breathing, so that he had to give up a treat which cost him too dear a price.

LECTURE XXII.

HOOPING-COUGH.

§ 1. Specific Pulmonary Catarrh.—It is contagious, and affects an individual only once in his life.—Incubation stage.—Period of invasion.—It begins like a common Cold, which occasionally presents special characters, and may sometimes entirely constitute the disease.—The fever of the invasion stage lasts from seven to eight, ten, twelve, or fifteen days.—Stationary or convulsive stage.—Characteristic inspiration.—Expectoration of Bronchial Mucus.—Vomiting.—The paroxysms are more frequent at night than during the day.—Third period.—The whole duration of Hooping-Cough is limited with great difficulty, and is directly proportionate to the duration of the Prodromata.

Gentlemen,—As there are just now in the nurses' ward two children who are suffering from hooping-cough, I wish to give you a history of that complaint, and to describe it to you in such a way as to impress it deeply on your mind, and make the lesson practically useful to you.

You all know that hooping-cough is characterised by paroxysms which recur with greater or less frequency, and are more or less prolonged, consisting in several abrupt and jerking expiratory movements with loud coughing, followed by a long, painful, and whistling inspiration, which is somewhat pathognomonic.

The nature of this complaint has been regarded in various lights. Some considered it to be a neurosis, and others, a catarrh. It is both in reality, for the neurotic and catarrhal elements are always found. In my opinion, and in that of a great many physicians (and in particular of Dr. G. Sée, who has written a remarkable essay on the subject), hooping-cough is a special affection, a specific pulmonary catarrh. I say that it is a catarrh, because, as I told you just now, the catarrhal element is invariably present, and it is therefore a character which should serve to designate the kind of disease. The nervous element which is superadded to it, the nervous phenomena which accompany it, and which belong to hooping-cough exclusively, impart to it the specific character which will strike you in all that I shall say of its causes, its mode of transmission, its progress, its duration,—of its symptoms, in a word.

In the first place, this complaint may not only be epidemic, but it manifestly is also highly *contagious*. The fact is admitted by everybody. Now, as I have stated to you on several occasions, whenever a disease is transmissible from one human

individual to another, from the lower animals to man, or from the latter to the lower animals, the fact necessarily implies the idea of speficity. For there can be no contagion without a germ of a special nature, capable of developing itself in a fit soil, and of reproducing itself, and manifesting its influence by constantly identical phenomena. This, then, constitutes already an important character, and this would of itself suffice to rank hooping-cough in the very extensive class of specific diseases.

Like most specific disorders, *it commonly attacks the same individual but once in his life.* There are yet exceptions to this rule, and I have myself known two cases in which children have had hooping-cough twice. Why, after all, should it behave differently from syphilis, or from eruptive fevers, such as variola, measles, scarlatina, typhoid fever, which have been known to attack the same individual several times, although they do not, as a rule, return?

Like specific diseases, again, hooping-cough is *chiefly met with in children;* and when adults, and even old people, suffer from it, it is either because they had escaped the disease at an earlier period of life, or because they are attacked a second time, as in the rare instances to which I alluded just now.

Lastly, this disorder has an *incubation stage*, which cannot, it is true, be defined, but which cannot be called in question when the fact is taken into account that hooping-cough never shows itself immediately after exposure to contagion, and that a certain number of days elapses before symptoms of the disease manifest themselves.

More than five-and-twenty years ago, I was summoned to an hotel in Chaussée-d'Antin Street, to see a young lady who had just come from Bordeaux, and who, according to her father's statement, had caught a violent cold on the journey. She was intensely feverish when she reached Paris, and coughed incessantly night and day. Her cough had none of the characters of hysterical cough, of which I have had the opportunity of showing you examples here. It resembled the cough of a very acute catarrh, with this difference, however, that in ordinary bronchitis there are intervals of quiet, however short they may be, whilst in this case the cough was incessant, recurring twenty, thirty, forty times in the minute. There was, as I have said, very high fever. On ausculting the chest, I only heard some sonorous rhonchi. At first, I confess I thought that the case was one of galloping phthisis, and I could not conceal my anxiety from the patient's friends. But as a few days went by, the characters of the cough changed: there were eight or ten very violent shakings in succession, followed by a few minutes' rest. These characters soon became more distinctly marked, and came to resemble those of hooping-cough, so as to settle all

doubt on the point in my mind. By questioning the patient's friends, and inquiring into the previous circumstances of the case, I then learnt that a younger brother of this young lady, who had been left behind in Bordeaux, had had hooping-cough, which was at that time epidemic there. Had I been told of this circumstance on my first visit, I should have diagnosed the disease much more easily.

Enlightened by this case, I have since had the opportunity of observing several such; and both in private practice and in the children's wards placed under my care at the Necker and the Children's Hospitals, I have been enabled to recognise hooping-cough by the *obstinate coughing*. When I saw a patient with a cold which gave rise to paroxysms of coughing, recurring fifteen, twenty, and thirty times in the minute, and which continued in this manner for four, six, eight, or ten days in succession, attended with high fever, I at once recognised a specific catarrh, and after a certain time, varying between one and two weeks, hooping-cough manifested itself with its distinctly-marked characters.

On the contrary, in some cases (which are much rarer than the preceding, for I have only met with two instances of the kind), the nervous element may show itself alone. From the beginning, the child has *spasms in the throat*, a kind of hiccup which consists in an inspiratory laryngo-tracheal whistling, like the one which is heard at a later period, at each paroxysm of hooping-cough, and is quite pathognomonic of the disease. In one of the cases to which I alluded just now, I noted the curious circumstance that the paroxysms came on exclusively during expiration. Thus, the child had three or four inspiratory coughs, accompanied by a whistling noise, and expiration was not modified in the least; then, a few days later, this whistling was preceded by efforts to cough, which then occurred during expiration, and in a short time hooping-cough assumed its usual course and symptoms.

In the great majority of cases, I repeat, hooping-cough begins like a simple catarrh, both in children and adults. There is this distinction, however, that the cough is somewhat more frequent and obstinate, and that the patient has a more troublesome sensation of tickling in the throat and inside the trachea.

This catarrhal cough lasts from three days to a fortnight, sometimes three weeks, a month, and even longer, before it assumes the specific characters which it will manifest at a later period. I have in some cases known it continue throughout the complaint, and no convulsive cough supervene. Will it be denied that such cases were really instances of hooping-cough? But notwithstanding the absence of the specific cough, the

other manifestations of the disease were amply sufficient for making a diagnosis. The catarrh was, in such cases, unusually obstinate. Thus, the patient's former colds had got well after a week or two, whilst this present attack lasted two, three, or four months. This cold had been caught at the same time as his brothers, sisters, or other companions became affected with hooping-cough. Like them, he had shown, in the beginning, *febrile symptoms*, which had lasted for three, four, five, six, eight, or ten days. The characters of the expectoration had been the same in all, and vomiting had come on in all after a fit of coughing. All the symptoms were present in such cases, therefore, except the convulsive character of the cough. Far, then, from calling in question the nature of the disease, such instances afford a new proof of its analogy to specific disorders, and to eruptive fevers in particular. In the latter we occasionally note the absence of an element which seems to be, and is indeed, their most important character, namely, the eruption, in measles and in scarlatina especially. Is the importance of the other symptoms to be called in question, then, when the specific eruption is absent? And why should it be otherwise with hooping-cough? The catarrh of the invasion stage is generally accompanied by *fever*, which is of greater intensity and longer duration than in a common cold. It rarely happens, as you are well aware, that unless the case be one of capillary bronchitis, whether the patient be a child or an adult, the fever which ushers in an attack of simple bronchitis continues longer than forty-eight or seventy-two hours. In hooping-cough, on the contrary, it is very common to find the fever of invasion last seven, eight, ten, twelve, and occasionally even fifteen days. So that, when I told you just now that hooping-cough began like a simple cold, I should have added that the catarrh of the invasion stage has nothing in common with ordinary catarrh except the cough which accompanies it; it differs essentially from it in regard of the attendant phenomena; and this cough itself, as I pointed out, differs in certain respects from simple bronchitis. From its earliest appearance, therefore, the manifestations of hooping-cough indicate its specific character.

The second stage is that of *spasm*, or of *convulsive* cough; or, if you prefer, the period when the disease is fully developed.

Allow me, gentlemen, to make a short digression here about the etymology of the term *coqueluche* (hooping-cough). It has come down to us from the middle ages. According to some, the disease was called after the hood (*cucullio*), which was used as a head-covering for persons suffering from hooping-cough; but according to others (mentioned by Sprengel), the word *coqueluche* is derived from *coquelicot* (red poppy), because the syrup of red poppy was for the first time employed in medicine

in the treatment of hooping-cough. Others again say that it is derived from the word cock, from the hoop which occurs at the end of a fit of coughing, resembling the crowing of a young cock. The disease used to be and is still called in Picardy, the *cough which hoops*, whence the English name for the disorder, hooping-cough. Singular as these denominations may be, and however unscientific the term *coqueluche*, they have the immense advantage of being perfectly understood by everybody, and of at once presenting to the mind the idea of a special affection.

During the first stage of hooping-cough, the patient, as I have said before, coughs incessantly night and day. The cough becomes less frequent in the convulsive period; it returns every two minutes, for example, instead of every minute, while true paroxysms supervene with more frequent puffs of coughing; at first there was only one puff, but in proportion as the disease progresses, the number of these increases, so that even about the end of the catarrhal period, five or six might be counted; in the second stage, the patient has ten, twelve, fifteen, twenty, in succession without drawing in his breath. The characters of this cough are so very special, that it cannot be mistaken; there is nothing like it in any other catarrh, and hysterical nervous cough differs from it essentially.

When the patient is old enough to give an account of his sensations, he often complains of a pretty acute pain in front of the chest, of a sensation of tickling or pricking in the larnyx and trachea which incite him to cough. His attempts at resisting are vain, and only retard without ever averting, the paroxysm. The convulsive cough then bursts out as it were; whereas in a common cold or in any other affection of the respiratory passages in which cough exists, the patient can more or less easily draw in his breath after a few expiratory puffs; he is unable to do so in hooping-cough. An inspiratory movement first precedes the fit and is followed by a series of expiratory movements which at first succeed one another slowly, and recur, as I said just now, a great many times, driving out all the air contained within the chest and not allowing the patient time for breathing. The veins of the neck and face become turgid, the eyelids swell, and the eyes become injected with blood; tears are secreted in abundance; the cheeks and ears are congested, and this congestion spreads to the whole surface of the trunk, which is bathed in profuse perspiration. The unfortunate patient, whose respiration is so violently interfered with, falls into a fainting state, which is occasionally pushed as far as complete syncope. At length, the convulsive movements of the respiratory muscles grow calm, an inspiratory effort takes place, accompanied by the characteristic hoop, which

is perhaps due to spasmodic contraction of the larynx, the muscles of which have likewise been convulsed. This inspiration announces a moment's rest, but the truce is only of brief duration, and the same series of symptoms as before soon reproduce themselves. This second paroxysm again terminates in the same manner by an inspiration, which is this time longer than the first, and after several paroxysms of this kind, the patient looks exhausted with fatigue. In general, during these fits, which may last several minutes, he brings up a ropy, viscid, colourless liquid, to a considerable amount, and at the close he is sick and casts up mucus and food. There is pretty frequently also no vomiting. Sometimes, as in a recent case which came under my notice, that of the child of one of my pupils, the paroxysms terminate by a fit of sneezing or two.

When the paroxysms are very violent, they frequently cause accidents to which I shall call your particular attention, namely, epistaxis, subcutaneous hæmorrhages, bleeding from the mucous membranes, and hæmoptysis; also cerebral congestion, to which are to be in some measure ascribed the convulsions which occasionally carry the children off. But before I speak of these complications, let us enquire into the patient's condition during the paroxysm. A child, for instance, is at play; a few minutes before the fit comes on, he stops; gay just now, he becomes sad; if he happen to be with companions, he goes away from them, and tries to avoid them. The reason of this is, if I may be allowed the expression, that he then *meditates* his fit, and feels it coming, on account of the sensation of pricking, or tickling, of which I spoke just now. At first he tries to avert the paroxysm; instead of breathing naturally and dilating his lungs to the fullest extent as he did just now, he holds his breath, he seems to understand that by entering his larynx in a volume, the external air will excite the fatiguing cough of which he has a sad experience. But whatever he may do, I repeat, he will fail in averting the fit, and will merely succeed in delaying it. Crying, or emotion of any kind which excites his nervous system, quickens the paroxysm. When this has come on, you see the patient seek a fixed object near him of which he may lay hold. He rushes into the arms of his mother or of his nurse, if he be a baby; if he be of a more advanced age, and happen to be standing, he stamps his feet in a state of convulsive agitation. If he were lying down he sits up quickly, and clutches his bed-curtains or bed-posts. After the fit his face is swollen, and this turgidity of the face, which sometimes continues for three weeks, may sometimes suffice of itself to make an experienced practitioner suspect hooping-cough.

These paroxysmal attacks recur a variable number of times in the course of the twenty-four hours. It is a remarkable cir-

cumstance that they are commonly more frequent by night than by day, or to speak still more accurately, from six o'clock in the evening to six in the morning, than from six in the morning to six in the evening. Can one attempt to account for this difference by saying that at night the child is no longer, as during the day, under the influence of excitation which occupied his nervous system in another direction? Whether this explanation be true or not, the fact is no less true and deserves to be noticed. In some cases, however, the reverse obtains, and the child has more paroxysms during the day than at night.

Being desirous of ascertaining the number of paroxysms which a child might have in the course of twenty-four hours (as may be easily done), I adopted the following plan in my children's ward in the Necker Hospital. I asked the child's mother to prick, at each fit, a card with a pin, and by adding up the number of holes, I made out the next day how many paroxyms had occurred since my visit of the previous day. I was thus enabled to draw the conclusion, from a pretty good number of cases, that when the disease is of medium intensity, a child may have about twenty fits in the course of twenty-four hours; when it is more violent, he may have from forty to fifty, and in still more severe cases, the number of paroxyms may get up to sixty, eighty, a hundred even. When the number exceeds forty, the prognosis becomes grave, whence this proposition may be laid down that, under the same circumstances, the disease increases in danger in proportion to the number of paroxysms. More than this, it may be almost absolutely affirmed, that when the attacks return more than sixty times in the twenty-four hours, the child will die of some of the complications to which I have already alluded, and of which I am going presently to speak.

In the *third period* of the disease, the paroxysms become more and more rare, less and less prolonged, and less and less severe. The characteristic hoop of the closing inspiration is less and less marked, and then disappears entirely. Yet, when from any cause, from exposure to cold, or from mental emotion, the child, who had not coughed for several days, does so again, the paroxysms are exactly like those of the second stage. At this period of decline, the bronchial mucus which the patient expectorated after each paroxysm, is replaced by thick, opaque greenish, occasionally purulent sputa, having all the characters of genuine catarrhal expectoration.

The *duration of hooping-cough* is a point of the highest importance. Almost every day I call your particular attention to the absolute necessity of fully knowing the natural course of a disease. I have told you many a time here, and I will repeat it again and again, that this grave question is of paramount

importance in practical medicine, because it is, indeed, an accurate knowledge of the natural course of a disease which can alone enable us to estimate the value of the methods of treatment which we employ. Eruptive fevers, to which, as Dr. Sée judiciously observes, hooping-cough presents such striking analogies, and other diseases, like phlegmonous angina, for example, which develop themselves within a very distinctly-defined period, may prove fatal before the time appointed by nature, either from intercurrent complications, or from inopportune and unskilled interference of the medical attendant, but treatment never shortens their duration. Besides these diseases, with fixed periods, there are others, such as typhoid fever, the duration of which cannot be exactly known beforehand, although the evolution of their symptoms is equally unavoidable. Hooping-cough belongs to the latter class. It is most difficult to limit its duration rigorously. In some cases, it gets well in a week, sometimes in less, and I remember a child, a patient at the Necker Hospital, in which it lasted three days only. Hooping-cough was then epidemic in my wards, and nearly all the children in them had caught it. The child to whom I allude suddenly showed symptoms of a violent catarrh, which was the next day accompanied by repeated and characteristic convulsive paroxysms. These recurred for the next seventy-two hours, and on the fourth day there only remained the signs of an ordinary coryza. This boy remained for some time in my wards, and although, I repeat, hooping-cough was epidemic there, he never presented any other symptom of the disease.

One rarely meets with such fortunate cases in private practice. The evolution of hooping-cough takes most commonly six weeks at least, and the complaint lasts in general from fifty to sixty days. In opposition to the exceptional cases in which it gets well in a week and even less, others occur in which the disease lasts several months and even a whole year. In order to estimate the efficacy of any plan of treatment whatever in hooping-cough, this natural course of the disease must be therefore taken into account. No method of treatment should be regarded as useful until it has been tried in a great many cases, and has been found to cure in less than six weeks, or to diminish at least the frequency and the intensity of the paroxysms.

It is an interesting fact that the general duration of the disorder is directly proportionate to the duration of the prodromata. If these have lasted a short time only, hooping-cough is of short duration also, and the more quickly the convulsive cough has made its appearance, the more quickly also does it recede. So that, although there are pretty numerous exceptions to this rule, the ulterior course of an attack of hooping-cough

may to a certain extent be prejudged from the course of the disease at its onset.

§ 2. Complications. — Capillary Bronchitis. — Peripneumonic Catarrh. — Pleurisy.—Pulmonary Congestion.—Pulmonary Phthisis.—Vesicular and Interlobular Emphysema.—Vomiting.—Diarrhœa.—Hœmorrhages.—Rupture of the Tympanum, and Bleeding from the Ear.—Cerebral Congestion.—Convulsions.—Treatment.

The *complications* which supervene in the course of hooping-cough are of various kinds. Some of them are inherent in the very nature of the two principal elements of the disease, which is, in my opinion, a specific catarrh characterised by the nervous phenomena which I have described. Under certain circumstances, the catarrhal element becomes excessively acute and intense, and an inflammatory condition is set up which modifies the regular course of the disease, and gives rise to complications which may become dangerous.

When hooping-cough runs a regular course, there is only heard on ausculting the chest, when a paroxysm is impending, a weak respiratory murmur, probably on account of the spasm of the bronchi: loud sonorous mucous rhonchi are heard at the same time. When the paroxysm is over, the vesicular murmur is either normal or, as more commonly happens, a few large moist bubbling rhonchi, and sonorous ones, are still heard. When catarrhal complications arise, fever is lighted up; there is considerable oppression at the chest; on auscultation, fine mucous rhonchi, soon passing into the sub-crepitant kind, are heard, and then tubular breathing, signs of the *capillary bronchitis*, and of the *peri-pneumonic catarrh*, which have become developed. Sometimes also, especially in children and in adults, there are dulness, absence of all respiratory murmur, tubular breathing and ægophony, indicating the presence of *pleuritic effusion*. These inflammations of the lung substance and of the pleuræ are the most frequent causes of the patient's death.

The phenomena, which then manifest themselves, point again to the specific character of the disease. If, as some assert, hooping-cough were merely an intense form of bronchitis; and if the nervous manifestations of the convulsive cough were dependent upon the inflammatory element, these manifestations should be more exaggerated in proportion as the bronchitis became more acute, while, on the other hand, they should diminish or cease simultaneously with the symptoms of inflammation. But it is the reverse which occurs. Hence, when in a child suffering from hooping-cough, who used to have from 50 to 60 paroxysms in the course of twenty-four hours, you find these paroxysms cease suddenly, although the disease is still in the middle of the stationary stage, be on your guard and take care

not to augur favourably from this circumstance, because you will have to deal with some inflammatory complication; the convulsive phenomena, ceased so suddenly only because they have been silenced by fever; the nervous has been put down by the inflammatory element.

If a febrile affection manifest itself in the course of hooping-cough; if, for instance, the patient get measles, scarlatina, or small-pox; or if a phlegmonous inflammation supervene, accompanied by general reaction and fever, this fever, in the language of Hippocrates, stops the spasm, *spasmos febris accedens solvit*, and the phenomena dependent upon the nervous element cease for a while. If there be no eruptive fever, and no external inflammation to account for the febrile condition and for the cessation of the paroxysms, be on your guard, I repeat; auscult the chest carefully, and you will discover signs of capillary bronchitis, of peripneumonic catarrh, fine sub-crepitant rhonchi and tubular breathing; or signs of pleuritic effusion, tubular breathing, and ægophony.

The nervous element depends so little on the inflammatory element that, when you find the paroxysms recurring as frequently as before, you may predict beforehand that stethoscopic examination of the respiratory apparatus will point to retrocession of the inflammation.

The catarrhal and nervous elements of hooping-cough are, therefore, perfectly independent of one another. They run a parallel course when the disease is regular, but they separate when one of them, from some cause or another, becomes exaggerated and runs a different course from its usual one.

Do not think that this occurs in hooping-cough alone; for it is observed in other diseases, made up of compound elements, and I have called your attention to the same thing in spasmodic asthma. If in this latter complaint, bronchitis, pneumonia or pleurisy supervene, the paroxysms of dyspnœa cease, and although the patient's breathing is then more oppressed than that of other individuals, it is yet less so than previously, and in a different manner from what it was when the patient had fits of asthma. I told you to be on your guard when the spasmodic symptoms of hooping-cough disappear suddenly, because the inflammatory complications which silence them are more grave, other circumstances remaining the same, when they occur in the course of this disease than when they supervene in ordinary circumstances.

The efforts at expiration during the paroxysms necessarily producing a certain amount of pulmonary congestion, the capillary bronchitis, the pneumonia or pleurisy, will be all the more serious and will get well all the more slowly as hooping-cough may last 4, 5, 6 months and more, so that the return

of the paroxysms will prevent the complete disappearance of a congestive condition which the efforts at coughing tend to keep up and may aggravate.

As the persistence and obstinacy of inflammatory phenomena favours the evolution of diathetic manifestations, you may conceive how it happens that hooping-cough is such a frequent determining cause of the development of *pulmonary phthisis* in children with the tubercular diathesis; but I cannot admit, as a physician of undoubted talent does, that the specific character of hooping-cough has something to do with this. The inflammatory element alone seems to me to be accountable for the development of tubercles.

The air-cells of the lungs may burst in consequence of the violence of the paroxysms of coughing, and air may thus diffuse itself into the interlobular-cellular tissue. There is, then, in the interval between the paroxysms an extraordinary sense of oppression at the chest, and it occasionally happens that this *interlobular emphysema* spreads to the subcutaneous cellular tissue. The air diffused between the lobules of the lung makes its way along the roots of the bronchi, and gives rise to a subcutaneous emphysema which spreads more or less rapidly along the trachea, and first manifests itself in the cervical region which swells considerably and crepitates in a characteristic manner when pressed. This subcutaneous emphysema may affect the whole body by ascending along the trunk. Such an accident is grave, and generally terminates fatally. It is fortunately rare, but it yet deserves to be mentioned, although Rilliet and Barthez do not speak of it in their work on Diseases of Children as one of the complications of hooping-cough.

Vesicular pulmonary emphysema is nearly always found when the body of a child who has died of hooping-cough is examined. It is a necessary result of the violent coughing. After the complaint has got well, the air-cells gradually recover themselves, and all traces of the lesion disappear. The same thing happens in adults, when emphysema has been the consequence of a very obstinate attack of bronchitis, which at last gets well. But when hooping-cough attacks persons of advanced age, as in cases that have come under my observation, it produces irrecoverable vesicular enphysema, and when it is cured, the sense of oppression at the chest continues to the close of the patient's existence.

During the paroxysms, the patient often passes his *urine*, and even has involuntary evacuations, from the sphincters of the bladder and large intestine being unable to resist the violent efforts of coughing. *Hernia* is also frequently produced in hooping-cough as a consequence of these efforts.

The same cause (namely, energetic and convulsive contraction during effort) has been ascribed for the vomiting, which, as I have told you already, follows upon each paroxysm.

But we have seen that this accident could be regarded as inherent in hooping-cough. It seems to constitute its natural crisis, so much so, that a paroxysm of hooping-cough, whatever be the number of the fits of coughing, usually terminates only when vomiting has taken place. It is therefore a very common phenomenon, and is occasionally attended with grave consequences. Say, for instance, that a child has a good many fits of coughing in the course of the 24 hours, say 30 or 40; which consequently recur about every half-hour, as every one of them is followed by vomiting, the child's nutrition must necessarily suffer as he brings up all the food he takes. When the medical attendant, therefore, does not take care to carry on the treatment, according to the plan which I shall presently describe to you, with the view of combating this fearful complication, it is not rare to find this obstinate vomiting carry off the child, who literally starves to death.

The disorders of nutrition, by depriving the blood of its materials for repair, have probably something to do with the production of *hæmorrhage*, to which a patient suffering from hooping-cough is liable, although the obstruction to the venous circulation is in some measure sufficient to explain them. The blood-vessels get congested during the efforts at coughing, and this congestion which is transient at first, becomes at last permanent from being constantly repeated, and may be carried to such a point that the blood itself, or its more liquid parts, may escape from the capillaries.

Epistaxis is the form of hæmorrhage which most frequently occurs, and one may pretty often see a child bleed from the nose in a fit of hooping-cough. When this accident does not recur frequently, it presents no gravity, but not so when epistaxis occurs from the outset of the complaint, and is somewhat copious, and returns regularly. In the beginning, as the blood is of normal plasticity, hæmorrhage only occurs when there is vascular congestion; when the circulation resumes its normal course, the bleeding ceases; but when, in consequence of the repeated hæmorrhage, the blood has become less plastic, epistaxis occurs not only while the face is congested, but also continues for some time afterwards. As the plasticity of the blood goes on diminishing, and the child becomes more and more anæmic, he bleeds more and more abundantly from the nose, and the epistaxis is so prolonged that medical interference is needed to stop it. You may conceive what very serious complications these hæmorrhages are, not because I believe that they often cause the patient's death, but because they render him liable

to nervous accidents, to convulsions, which are never so common as in children who are exhausted by loss of blood.

Blood may also be *expectorated*; in some cases this blood comes from the mucous membrane of the mouth, from the gums, the pharynx, the posterior nares, but in others, it comes from the surface of the bronchi. *Hæmoptysis* is a pretty common accident, although the reverse has been asserted; some authors have gone so far as to state that, when moderate, it was a favourable symptom. Without concurring in that view, I admit that hæmoptysis is, generally speaking, of no gravity whatever, and need cause no anxiety.

In the rapid sketch which I gave you of a paroxysm of hooping-cough, I stated that, under the influence of the violent efforts at coughing, the face got congested, the eyes injected with blood, and tears were abundantly secreted. I will now add that the blood-vessels of the eye may be congested to such a degree as to give rise to conjunctival hæmorrhage, and I have seen a little boy, two years of age, who was suffering from severe hooping-cough, cry *tears of blood.*

In the case of a young woman, small drops of blood came out during each fit of coughing from a *nævus maternus* situated underneath her left eye. This singular form of hæmorrhage continued all the time that the hooping-cough lasted, and yet the disease was very mild.

This tendency to hæmorrhage often gives rise to *subcutaneous ecchymoses.* In a little girl from nine to ten years old, there occurred during a severe attack of hooping-cough, an extravasation of blood into the *subconjunctival* cellular tissue, and into the cellular tissue of the eyelids, which passed through the ordinary stages of resolution, and successively stained the affected parts of a dark red, a violet red, a brown, and a greenish-yellow colour. You will, doubtless, meet with similar cases.

Hæmorrhage from the ears is a rarer accident, and two instances of it came under Mr. Triquet's notice at his dispensary, in the winter of 1860. The children's mother had noticed a flow of blood from their ears during a fit of coughing in the course of hooping-cough. On examining the auditory meatus and the membrana tympani, a linear rent of the latter was found a little below the handle of the malleus. In both instances the left membrane was alone torn.

One of the children was six, and the other five years old. In England Dr. Gibb has met with this accident four times, in children from four to nine years old.[1]

These four cases occurred in the course of an epidemic of

[1] British Med. Journal, and London Gazette, November, 1861.

hooping-cough, which had attacked 200 children from six to nine years old.

Now, in these four children, and in the two which came under Mr. Triquet's observation, an examination of the ear always detected a linear rent of the membrana tympani.

In two of Dr. Gibb's patients, the membrana tympani of both ears was ruptured, and in one of the children the rent was triangular or heart-shaped.

In four of the eight cases the seat of the rupture was near the circumference of the membrane, in two it was central, and in one the membrane was torn into three pieces, from one to two millimetres in length. A small coagulum, which intervened between the lips of these small rents, pointed in a positive manner to the source of the hæmorrhage, namely, laceration of the mucous or inner layer of the membrana tympani. The rupture in every case, except where the membrane was torn into three pieces, healed up by immediate or primary adhesion in the space of a few days. The triangular rent was the only one which did not get well, and it gave rise to prolonged suppuration and obstinate deafness.

The mechanism of such an accident is easily understood. During the efforts and convulsive coughing of hooping-cough, air is violently driven through the Eustachian tube into the cavity of the tympanum. The pressure exerted by the column of air overcomes the resistance of the membrana tympani, lacerates it in its weakest part, beneath the handle of the malleus, or tears it away at its circumference; and the laceration of the mucous lining of the membrana tympani gives rise to the hæmorrhage which comes from the ear, in rare but undoubted cases, as shown by actual examination. From these hæmorrhagic accidents, I am naturally led to speak of the *convulsions*, which, as I said just now, are often their indirect consequence, when very copious and frequent loss of blood has thrown the patient into a state of anæmia, which gives rise to a peculiar nervous susceptibility.

Attacks of eclampsia may again be a direct consequence, if not of the hæmorrhage itself, at least of the causes under the influence of which the hæmorrhage occurred. They are, perhaps, then caused by cerebral congestion, and seem to be dependent upon a peculiar modification of the cerebral centre, brought on by the afflux of blood resulting from the fits of coughing.

You must all be familiar with the sense of vacancy, of astonishment, which comes on after a violent effort kept up for a little while, and which is evidently the consequence of a transient congestion of the brain. This phenomenon of effort recurs in hooping-cough at very short intervals, and at last gives rise to more serious accidents. Thus, when the patient

is old enough to describe his sensations, he often complains after violent fits of coughing, of *headache*, which is occasionally so intense as to make him cry. This pain in the head is followed by a state of hebetude, like what is caused by concussion of the brain, and which lasts more or less. In some cases, symptoms of true cerebral congestion show themselves. I once attended a lady who used to fall into the same state of stupor as that which follows epileptic fits; this lady suffered also on several occasions from incipient paralysis, marked weakness of one arm. You understand how such a disturbance of the functions of the brain may, in children, give rise to convulsions.

Convulsions may come on also independently of hæmorrhages and of congestion. They are then due to the nervous element which gives to hooping-cough its specific character; the nervous excitability which already manifests itself, as a rule, by the production of convulsive fits of coughing, spreads to the whole system, either in consequence of the weakness of the patient's constitution, or of acquired weakness, his strength having been exhausted by the prolonged duration of the complaint, by disordered nutrition, or any other cause.

These nervous accidents, which sometimes also consist in *delirium*, in *extreme agitation*, are more frequent and more grave in proportion to the tender age of the child. They nearly always terminate fatally when they coincide with the inflammatory complications which I mentioned to you.

The *treatment* of hooping-cough is a matter of considerable difficulty, because it is a complaint which yields with extreme difficulty to the various measures which we can oppose to it. Yet I do not admit that we are entirely powerless against it; and, in opposition to J. Frank, who asserted that we can kill a patient suffering from hooping-cough before the term of his complaint, but that we can never cure him, I believe that in a pretty good number of instances, a well conceived plan of treatment markedly diminishes its duration.

I will not pass in review the various remedies which have been lauded against it. Every author has his own formula, and I need not enumerate all these pretended specifics. I will merely indicate to you a few of the methods of treatment which appear to be of some utility, and will dwell specially on the one which is, in my opinion, of unquestionable efficacy.

According to Laennec,[1] the most useful treatment at the outset of hooping-cough, consists in the administration of *emetics*, repeated every day, or every other day, for a week or two. Children, after all, bear vomiting much better than adults. Laennec even preferred, in the case of children, *tartar emetic* to

[1] Traité d'auscultation médiate, 4th ed. Paris, 1837, t. iv., p. 228.

ipecacuanha, because of the extreme variability of strength of the different kinds of ipecacuanha met with in commerce, and which are obtained from different plants. Tartar emetic, he added, being soluble, may be much more easily administered in as small doses as may be required by the age and feebleness of the child. Others, instead of tartar emetic and ipecacuanha, prefer *sulphate of zinc* or *sulphate of copper*, on the ground that they have an antispasmodic, in addition to their emetic, action.

I do not admit this twofold action of the salts of zinc and copper. When I wish to bring on vomiting in a child, I, however, give sulphate of copper, because it is the most certain emetic which I know of. I prefer it to ipecacuanha, because, as Laennec remarked, ipecacuanha often fails, and to tartar emetic, because this latter sometimes gives rise to very unpleasant consequences. However prudently it may be administered, according to the individual, and according to his condition at the time, it may act more powerfully than was intended. It has in some cases brought on profuse purging, vomiting, and diarrhœa, and has given rise to choleriform symptoms, and thrown the patient into a truly alarming state of weakness.

For these reasons I prefer using the sulphate of copper, which I administer after the following method:—I order from five to nine grains of the salt in the case of a child, and fifteen grains in that of an adult, to be dissolved in three ounces of distilled water, of which a dessert-spoonful is taken every ten minutes, until vomiting is produced.

This method of administering emetics in fractional doses is the one which you see me adopt constantly, whatever be the drug I prescribe, and whatever be the indications of the emetic treatment. By this means, I need not fear to go beyond the mark which I propose to myself. At the outset of hooping-cough, and when the disease is in its fully developed stage, when the cough is accompanied by symptoms of impending suffocation, emetics are of some utility, and I have known them in several cases very markedly diminish the number of paroxysms.

Antispasmodics must necessarily have held an important position in the treatment of a disease in which the nervous element plays a very marked part. Hence a great many formulæ contain valerian, castoreum, musk, assafœtida, ammoniacum, oxide of zinc, etc.; but the utility of these various remedies, and of antispasmodics in general, has always seemed very questionable to me.

Narcotics and *stupefying* drugs are much more efficacious, and of them, *belladonna*, to which I alluded just now, or its alkaloid *atropia* is, in my opinion, the most heroic remedy which can be used in the treatment of hooping-cough.

In order, however, that belladonna should produce its full effects, it should be administered according to a particular method, which is of such importance that if you neglect to observe it, you will not succeed in curing hooping-cough, any more than you will succeed in curing ague, however large the dose of bark you may give, unless you follow certain rules which I will some day point out to you.

Before I give you the details of the plan of treatment to adopt, I must first establish the following capital point:—The active principle of solanaceous plants influences neuroses only when given in sufficiently large doses, and this influence lasts for some time; but lest the therapeutic effects should be greater than desired, the medicine should first be given in doses which are probably less than those needed for exerting a favourable action on the disease; these doses must be gradually increased until therapeutic effects begin to show themselves. As soon as this result is obtained, it is generally sufficient to continue the same daily dose in order to increase the good effect produced. If the dose which has brought on these good results were increased hastily with the view of accelerating the cure, and especially if it were repeated on the same day, one might at first wonder at the success obtained, but an unpleasant dryness of the fauces, and some disturbance of vision, which increases rapidly, would soon render a diminution of the dose necessary, and the consequence of this would be to allow the disease to reproduce itself, and to escape the influence of the mode of treatment.

Bearing well in mind these general principles, the treatment is to be carried on after the following method:—

If the patient be an infant, have pills made containing each one-tenth of a grain of extract of belladonna, and one-tenth of a grain of powdered belladonna leaf. For children above four years old, and for adults, the pills should contain one-fifth of a grain of extract, and one-fifth of a grain of the pounded leaf. The pills should not be silvered; and as there are children who do not know how to swallow pills, even when they are given to them in jam, honey, or panada, they can be dissolved in a little syrup, and thus easily taken when put on the tongue. One pill is given in the morning, fasting, and another the following morning. First take care to ascertain the number of paroxysms by means of the method which I have told you, namely, by pricking a card with a pin, and keep a separate account of the diurnal and nocturnal attacks. You can then easily judge of the effects of the treatment by comparing the number of fits of the preceding day with that of the fits on the previous days. Thus, say that a child who had at first thirty-five paroxysms in the course of the twenty-four hours, has only thirty after taking belladonna, the medicine will evidently have acted on him. Or

say, that the sum total of the fits is the same, but that instead of there being four or five paroxysms, there be only two or three; or say again, that the paroxysms and the fits of coughing, of which they consist, are as numerous as before, but that the fits have been less violent: in all these cases there is, after all, real improvement, and the same dose is henceforth to be given. If, on the contrary, the fits have been as numerous and as violent, an additional pill is to be given, and two pills are to be taken at the same time. This is a point of capital importance, gentlemen. Whatever quantity of belladonna you may give, it is an essential point that it be taken in one dose. If you have been obliged to increase the original quantity ten or twelve times, let the patient take it in one dose, in the morning, fasting, at the same hour, and not at distant intervals in the course of the day. But before you increase the quantity, wait two or three days, and according as there has been an improvement or not, keep to the same dose, or increase it by degrees, unless symptoms of poisoning should come on, when of course you must stop.

When the fits of coughing have markedly diminished in number and violence, when, for example, they have fallen down from thirty to ten, go on giving for seven or eight days the dose of belladonna which has apparently brought on this improvement. If this continues, diminish the quantity of the medicine, by a reverse method to the former, namely, by taking off first one, then two, next three pills, and so on. If the fits of coughing increase again, resume the dose which stopped them. Lastly, when these fits have entirely ceased, and the disease may be justly regarded as cured, still go on with the belladonna for six or eight days before entirely stopping all treatment.

Since atropia has been used in medicine, it has been substituted for belladonna, and with this advantage, that while it possesses all the active properties of the plant, it has a fixed composition which the officinal preparations of belladonna do not always possess.

When the child is very young, I prescribe a solution of one-fifth of a grain of the neutral sulphate of atropia in five ounces of water, a tea-spoonful of which is therefore equivalent to $\frac{1}{200}$ of a grain of the alkaloid. This is the dose given in the beginning, and it is gradually increased, according to the rules which I laid down just now with respect to belladonna.

You must be on your guard against what I might term pseudo-relapses, for hooping-cough is a complaint which seems to recur after it has been really cured. A child may have, a month after the final cessation of the disease, a fit of coughing as in hooping-cough, when he cries or gets into a passion. More than this, the same thing may happen if he gets a catarrh, six or twelve

months afterwards. Do not from this infer that there is a relapse. His cough assumes those characters because his economy,—his nervous system—falls into its former bad habit, if I may be allowed the expression.

The treatment of hooping-cough by *revulsives*, by the application of blisters to the chest, frictions with croton oil, with spirits of turpentine, is far from giving the good results which has been ascribed to it. I would say nothing about it, if it were not to raise my voice against the dangers of a remedy about which so much noise was made by the man who proposed it: I allude to frictions with *Autenrieth's ointment*. Autenrieth ordered the chest of children suffering from hooping-cough, especially towards the close of the second stage, when the expectoration is becoming muco-purulent, to be rubbed three times a day with an ointment containing tartar emetic in variable proportions. This was to be continued, until pustules formed, which soon passed into ulcers. The appearance of these pustules, not only over the chest, but over other regions of the body also, especially the inner aspect of the thighs and the genital organs both in boys and girls, was in Autenrieth's opinion a sign that the system was saturated with the drug, a result which he thought one should always endeavour to bring about.

I have often told you my opinion with regard to this pretended saturation with tartar emetic, whether we look at its manifestations about the mouth or about the skin. The characteristic eruption to which it gives rise is said to be the result of the general effects of the drug, and not of the local irritation caused by tartar emetic when in contact with the skin or with mucous membranes. This eruption, however, is shown to be the effect of a local action by the fact that it does not manifest itself when the tartar emetic is given in pills instead of in a mixture, so as to prevent prolonged contact of the drug with the mucous membrane of the mouth and pharynx, and that when it is taken internally in very large doses, as in pneumonia, according to Rasori's method, no cutaneous eruption ever occurs.

Setting aside this pretended saturation of the system with tartar emetic, the treatment of hooping-cough by Autenrieth's ointment offers the greatest dangers and no advantage whatever. It is horribly painful, considerably more so than blisters, and occasionally gives rise to inflammation which, starting from the pustules, affects the cellular tissue, spreads in depth and gives rise to serious accidents. As one of several instances of the kind, I will mention the case recorded by Dr. Blache in the article on Hooping-cough in the 'Dictionnaire de Médecine.' The patient was a little girl six years old, and the use of the tartar emetic ointment produced the most lamentable results. Deep

ulcers followed upon the pustules, one of which, at the base of the sternum, was nearly two inches in diameter. It had exposed and completely detached from the bone the extremities of the costal cartilages, which floated in pus that was secreted in extreme abundance, in spite of all attempts at diminishing the suppuration. Symptoms of pyœmia soon showed themselves, and the child died of a colliquative diarrhœa which nothing could stop. Autenrieth was led into error by the circumstance that the convulsive phenomena of hooping-cough grow quieter after the use of the tartar emetic ointment, in consequence of the febrile reaction excited by the inflammation of the skin, but show themselves again as soon as the inflammation has ceased.

Besides these immediate disadvantages, tartar emetic ointment has others which are less serious, but are still worthy of the practitioner's attention. The pustules and the ulcers which form after them, leave behind indelible scars which may simulate the marks of scrofula. Trifling as this circumstance may seem, you will understand its value, and you will one day appreciate the utility of all these small details into which I am not afraid of entering in the course of my clinical teaching, whenever an opportunity presents itself.

Before concluding, I have to speak of the treatment of the complications of hooping-cough.

I have told you that *vomiting* was often a very serious complication of the disease, as it was apt in some cases to cause death by starvation. It is indispensable, then, that you should know how to feed the patient, and the first rule to bear in mind is to give him food so that he may keep it. Experience can alone enlighten you on this point; for some patients are sick only during the day, and you must then tell them to postpone their meals until evening. When vomiting recurs both during the day and at night, the patient should take food immediately after a paroxysm, because the next paroxysm will be some time before coming on. However averse the child may be to taking food, he must be made to do so, and he should have solids instead of liquids, as they are less easily brought up.

When belladonna or atropia is administered, the paroxysms return at longer intervals under the influence of that treatment. Advantage may be taken of these intervals of rest to give the patient nourishment; besides, even though it should not lengthen the intervals between the paroxysms, belladonna stops the vomiting or renders it less violent. In some exceptional cases, in spite of the administration of this remedy, the patient cannot keep down his food. Opiates should then be given in small doses, in combination with preparations of belladonna. The child should take immediately after vomiting, and just before eating, *half a drop, or even one drop, of laudanum*

(*of Sydenham*). To compel the patient to eat immediately after he has brought up his food and to give opium internally, are therapeutic stratagems of the greatest importance.

On account of the consequences which may follow it, *hæmorrhage*, especially when from the nose, (the gravest form of bleeding in hooping-cough) should be treated at once. Of the means that we may employ there is one the good results of which are real although unaccountable, namely, the plan of raising the arm which is on the same side as the nostril from which the blood issues. I might enumerate a great many hæmostatic remedies: astringent powders and liquids; injections with water acidulated with sulphuric, nitric, and hydrochloric acids; cold applications to the forehead and nucha; and various other measures which you know, and at the head of which I place injections into the nostril of water as hot as the patient can bear. In extreme cases, and when the bleeding resists every other measure, the nostrils may be plugged, as a last resource, either with Gariel's india-rubber bladders, or by means of Belloc's sound. Plugging of the nostrils presents no inconvenience in adults, but it may in children bring on excessive agitation which increases the violence and number of the paroxysms of hooping-cough. It should therefore be employed only as a very last resource. While acting directly on the seat of the hæmorrhage, internal remedies may be administered at the same time, such as acidulated drinks, the sulphuric lemonade, mixtures containing the *eau de Rabel*,[1] ratanhia, matico, kino, and best of all, powdered bark.

As to the grave inflammatory complications about the chest, such as capillary bronchitis, pneumonia, pleurisy, they demand special treatment of which I need not speak here.

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[1] [*Eau de Rabel* is a mixture of one part of sulphuric acid to three of alcohol.—Ed.]

LECTURE XXIII.

ON HYDROPHOBIA.

Nervous Phenomena characteristic of Hydrophobia.—Sensory Hyperæsthesia.—Priapism, a frequent symptom.—The Manifestation of *Lyssi*, during the stage of incubation, is not at variance with the pathology of virulent diseases.—They might be the primary localisation of the Virus.—Can Hydrophobia be prevented by cauterising them?—Analogies and differences between Human and Canine Rabies.—Rabies never spontaneous in man.—Treatment as varied as powerless.

Gentlemen,—At one of our last meetings, I called your attention to the case of a man who exhibited all the symptoms of hydrophobia, and who died on the same day that he was admitted into the hospital. From the information we received from his friends, and which was confirmed by Dr. Bienfait (of Rheims), and by Messrs. Leblanc, veterinary surgeons in Paris, no doubt could exist as to the nature of his complaint.

I know of nothing, gentlemen, more painful to hear, or more fatiguing to read, than a lengthy medical case overloaded with details; and yet, details should not be neglected, when they relate to a complaint which you will rarely have an opportunity of observing. A complete case, better than a long dogmatic treatise, leaves on the mind an impression which time cannot blot out, especially when you have been an eye witness of the chief phenomena of the disease. You will, therefore, forgive my entering into details which may at first sight appear superfluous, but will by and by be perfectly explained.

The case is as follows: During the night of January 23, 1861, my clinical assistant, Dr. Dumontpallier, was summoned in all haste to see a patient who was suffering from what was termed "an *indigestion of water*." On his way there, Dr. Dumontpallier was told that the patient complained of intense thirst and was firmly bent on drinking, but could not carry water to his lips without being seized with a deep feeling of terror. He could not take any solid food either. This difficulty of swallowing, supervening suddenly after slight *malaise*, in a man aged 37, was a strange phenomenon. By closely questioning the patient's friends, he then ascertained that about the end of September, that is, four months previously, M. B. had been bitten in the hand by a small pet dog, which had on the same occasion bitten a little girl, 8 years old, and a man-servant about 30 years of age, and a young cat, with which he usually played. It was added that the dog had died of inflammation of the bowels

in Mr. Lablanc's infirmary, in the beginning of October 1860. Mr. B.'s friends were honest in giving this information, because Mr. Lablanc had thought proper to conceal the name of the dog's real complaint. As the child, the servant, and the cat that had been bitten were perfectly well at the end of January 1861, nothing had occurred to create any alarm in Mr. B.'s family. The rapid death of the dog, however, a few days after he had bitten his master, and the complete dysphagia which had supervened in the latter, inclined Dr. Dumontpellier to think that the patient was most probably hydrophobic. When he saw him, he was walking about his room, in the greatest agitation, unable to remain quiet a single moment. He looked fixedly before him; his pupils were dilated, his face extremely pale, and his hair and beard disordered. The expression of his physiognomy was that of great anxiety; he spoke in a curt and jerked manner, and complained of great dryness of the throat and of his being obliged to keep spitting incessantly. Whenever he spat out the saliva, his whole body shivered. The room was lighted up with candles and a lamp; over the mantelpiece was a looking-glass and on the shelf a water jug. As the sight of these objects gave the patient no pain, there was, therefore, no hyperæsthesia of the eye, but the skin was painfully sensitive. The patient dreaded to touch his face and to rest his hands on his clothes; he refused to allow his pulse to be felt, and in order to shorten the examination, he wished by taking a glass full of water to show that he could not drink, although he had a firm intention of doing so, for he took up the glass and raised it to his lips, but he immediately rejected the water which, by a rapid movement, he had got into his mouth. This voluntary experiment brought on no convulsions; the patient was merely more agitated for a few minutes, and then having calmed down, he tried to relate what he had felt since January 20.

While speaking, he made prodigious efforts for remaining calm. He had been sad for a long time, he said, in consequence of money losses, and had gone to Rheims on January 13, to stay with some friends, in hopes that he might be cheered up. From January 13 to January 20, he had felt no *malaise*; at that last date—a Sunday—he went out early in the morning in an open carriage, the temperature being cold, on an excursion into the country with his friends. He ate in the morning with his usual appetite, but in the afternoon he felt so intensely thirsty that the carriage had to be stopped several times to allow him to drink at some houses on the road. He had then no difficulty in swallowing, but what he drank felt very cold to him. He was shortly afterwards seized with violent shiverings in the carriage, and went to bed immediately

on getting back to Rheims. He did not sleep at all during the night; he got up constantly because he felt giddy when lying down, and walked about in his room, feeling very agitated. He did not feel hungry, and he could still drink, although he had strange unpleasant sensations. He was very agitated during the whole of Monday, both during the day and at night. Mr. B. himself gave all these details, for his mind was perfectly clear. Dr. Bienfait (of Rheims) kindly wrote me two letters containing a description of the symptoms which he had observed from January 21, when he was first called in. I will read to you what he said:—

"The patient was in a state of considerable agitation; his face was pale and his eyes extraordinarily mobile. His own idea was that he was suffering from an overloaded stomach and that he was going to be sick. His breathing and the action of his heart were somewhat hurried. The tongue had a slight yellowish coating of fur, and was of a somewhat brighter tint along the edges and the raphe. The patient drank in my presence, but with a certain degree of convulsive haste, as everything else he did."

Dr. Bienfait thought that the case was one of indigestion attended with a nervous condition depending on the patient's idiosyncrasy; he prescribed a mixture containing opium. He was, however, uneasy on account of that nervous condition, and therefore went back to see him in the evening. He found the patient in a still more agitated state; he had taken his mixture, but each time after heroic efforts. "I asked him to take a spoonful of it in my presence," Dr. Bienfait goes on to state; "but it was immediately thrown out by a sudden spasm which seemed to spread from the muscles of the pharynx to the orbicularis oris. Yet the unfortunate patient had collected all his strength before attempting to swallow; he had taken three steps backwards, and by an instinctive movement, had thrown his arms about him as if he wished to get all obstacles out of the way." A bath was ordered, to the patient's great joy, but was not taken. Dr. Bienfait no longer entertained any doubt as to the nature of the case, and although he knew nothing of the previous history, he believed the patient to be suffering from hydrophobia.

On the following morning, the symptoms had become aggravated, and there was, moreover, general hyperæsthesia. The patient's friends, better informed, it would appear, than his own relatives, then told Dr. Bienfait that, about the month of September, Mr. B. had been obliged to sacrifice a small king-Charles, who was suffering, according to a veterinary surgeon's account, from rabies. Mr. B. was probably aware of

the circumstance, but never made any allusion himself to his dog having been mad. Nothing had been done to try and avoid ill consequences from the bite.

In his second letter, Dr. Bienfait stated that previous to allowing Mr. B. to return to Paris "he had, on ausculting his chest, found that the vesicular breathing was perfectly pure, but was interrupted at each inspiration by one or more suppressed sobs, as it were. The heart's impulse was notably irregular, and the pulse at the wrist was equally so; the irregularity of the latter being attended with a sort of vascular spasm, which could not be defined, but was very remarkable."

"During the whole of his stay at Rheims, the patient had no delirium, or anything like it, and never had the least wish to bite. He only spoke of some instinctive dread, and had a marked tendency to be communicative. He had no idea of the real nature of his complaint, and had no recollection of having been bitten. Imagination, therefore, seemed to have nothing to do with the manifestation of the symptoms recorded."

Mr. B. insisited on returning to Paris. During the journey from Rheims to Paris, he was very agitated and extremely thirsty, his thirst being temporarily relieved only by keeping small lumps of ice in his mouth; but in all probability the patient, who was constantly spitting, could not swallow the melted ice, and therefore complained of a sensation of constriction and great heat in the throat. He had, during the journey, frequent erections and seminal emissions: the hyperæsthesia of his genital organs gave him great pain.

Such was his condition when Mr. B. reached Paris in the evening. I have already told you in what state he was seen by my clinical assistant. The latter advised his immediate removal to the Hôtel-Dieu, and on the following morning, January 24, that is, $3\frac{1}{2}$ days after the invasion of the disease, I had the opportunity of ascertaining with my regretted colleague, Legroux, the following facts:—

The patient was so agitated that many thought him to be suffering from acute mania. He had a strange aspect and was unable to swallow liquids. Several among you, doubtless, remember the painful scene when he attempted to drink: he clutched the glass with force, saying: "I wish to drink, and I will," and then carried it with determination to his lips. But as soon as the water had passed his lips, his face assumed a look of excessive pain, and his whole body was within a short time shaken with violent convulsive trembling. He then exclaimed: "I cannot drink, do make me drink."

When calm had returned after this paroxysm, I was enabled to ascertain that there was redness of the soft palate and

pharynx, and great dryness of the tongue. The patient's beard was also soiled with a frothy saliva which he kept constantly spitting out.

The lateral and under surfaces of the tongue were carefully examined; the patient was docile, and had no desire to bite; but we could not discover any of those swellings which have been called *lyssi*. We could not, however, draw any conclusion from their absence, because we shall see, by and by, that they have been seen only during the period of incubation by the very persons who described them, and that they disappear before the symptoms of confirmed rabies show themselves.

I merely recommended that the patient should be watched, and nothing else, as experience had taught me that we are completely powerless against this cruel malady.

In the course of the day, the patient's wife and some of his friends came to visit him; he was always very agitated, and the presence of his friends gave him pain. He begged that everything should be tried to cure him: he would be saved, he said, if he could be made to drink.

About half past four in the afternoon, my clinical assistant, with the aid of several pupils, succeeded in introducing an œsophagus tube through the nares into the stomach, and about seven ounces of broth were gently poured into it. Half of this quantity had already reached the stomach, when the fluid suddenly ceased to run, from the flexible tube being compressed by a violent spasm of the pharynx and œsophagus. The spasm soon spread to the respiratory muscles, the face turned livid, and the opened eyes stared: the tube was quickly removed, and as the patient, who had been sitting on a chair, slipped down on the floor like an inert mass, it was thought that he had died; but nevertheless water was sprinkled on his face, his tongue was drawn out of the mouth, which was kept open by firmly drawing down the lower jaw, while the thoracic walls were alternately squeezed and left to expand. A whistling inspiration then followed; compression of the chest was kept up, and respiration was soon re-established, after which the patient ejected to some distance a certain quantity of saliva or of bronchial froth. During the paroxysm there had been an erection of the penis, with ejaculation. The patient evinced no fear on finding himself lying on the ground; he knew that he had just run a great danger, but he believed that he was saved. Advantage was taken of his being calm to advise him to get into bed, and he was persuaded to allow himself to be tied in it lest he should fall out. He let us do what we liked with him, and warmly expressed his gratitude; he squeezed our hands, and wished to kiss those who had saved his life, he said. In the course of the evening he had several convulsive paroxysms, and died suddenly

at half-past ten, after having been violently agitated for a few seconds.

A post-mortem examination was made on the very next morning. The body was exceedingly rigid, the face livid, and the whole posterior aspect of the trunk and limbs presented numerous sugillations. The brain and parenchymatous organs were congested. The mucous membrane of the mouth and pharynx were very markedly injected. The salivary glands were taken out and sent to Alfort, with some saliva that had been collected during the patient's life. Mr. Reynal, who had undertaken to inoculate dogs with this saliva, afterwards informed me that the experiment had given no result.

Note, gentlemen, that the child and the man-servant, who had been bitten, did not exhibit any symptom of rabies when Mr. B. died. The cat also, which had been bitten at the same time, was still in the house, and there had occurred no change in its habits which authorised a suspicion that it had been inoculated with the virus; yet, it was thought prudent to sacrifice it on that same evening.

If we now sum up the principal facts in this case, we find that a man is bitten by a dog in September, 1860. A little girl and another man are bitten on the same occasion, as well as a cat, by the same dog, and at the end of January, 1861, the master of the house, the person first bitten, alone manifests symptoms of rabies. Before that date, Mr. B. was sad, but this was ascribed to money losses. He leaves Paris in search of recreation, when suddenly, a few hours after a breakfast eaten with relish, he complains of very intense thirst. He soon is seized with general shivering, and from that moment he loses his appetite, and the capacity of swallowing liquids, while he becomes extremely agitated. He goes on in that way for about thirty-six hours. General hyperæsthesia is then noted, and from that time he cannot wash his hands or face, the least attempt of the kind immediately bringing on great agitation and violent shivering, and the same thing occurs when he tries to comb his hair or beard; he dreads touching his person with his own hands. It is probable that the hyperæsthesia which existed then determined, through a reflex action on the least contact of the skin, convulsions which assumed the form of general shiverings. There were rare intervals of quiet. To the cutaneous hyperæsthesia there is then superadded a very acute and frequently repeated excitation of the genital organs, and frequent erection, attended with seminal emission, increases the patient's agitation. Three days after the invasion of the disease, his aspect creates alarm; he is agitated, extremely garrulous, speaking in a curt, jerking manner; he cannot drink, although he is intensely thirsty, and when he attempts to doze, he is

immediately seized with clonic convulsions and spasms. The hyperæsthesia and satyriasis persist throughout the 24th of January, the convulsive paroxysms become more frequent, and death occurs on the fourth day of the complaint, without the patient having manifested any mental disorder, any hallucination of sight or hearing.

We could not succeed in finding the least trace of a bite on Mr. B.'s hands, although his symptoms did not admit of a doubt as to the nature of his malady, and the dog that had bitten him had likewise died of hydrophobia. Two periods were observed in Mr. B.'s case; one of sadness and melancholy, during which he left his house and went away from Paris in search of amusement; the other, which may be termed period of *agitation*, and which set in with very intense thirst and shivering, soon followed by a dread of water and by convulsions, which continued until his last moments. It is important to note that, as well as general hyperæsthesia, there was satyriasis present, a fact rarely mentioned in records of cases of rabies, as in the numerous instances contained in Ch. Andry's [1] work, and in the article by Trolliet and Villermé, in the *Dictionary*, in sixty vols. Boerhaave, however, mentions priapism as one of the symptoms of rabies in man,[2] and Van Swieten [3] states in his *Commentaries*, that Galen has mentioned the fact, which has also been noted by other observers. He relates the case of a porter who became hydrophobic, and who, during the last three days of the complaint, had involuntary and constant seminal emissions. This man, Van Swieten adds, lost at the same time his seed and his life: *Semen et animam simul efflavit.*

Dr. Peter has also noted frequent seminal emissions in the case of a soldier, who was admitted, in 1862, into the military hospital of Gros-Caillou, under Dr. Worms. The priapism was almost constant, and the emissions were attended with voluptuous sensations, as showed by the prurient expressions used by the patient. Shortly afterwards, in his delirium, he bitterly reproached his medical attendants for having recourse to witchcraft in order thus 'to take away from him the very principle of life.' He had been bitten forty days previously by a very small dog, which had entered the barrack room and did not look ill. The disease had set in with a fearful spasm, which occurred as he was going to wash his face. When he was brought to the hospital, he was perfectly rational, but carefully avoided all allusion to the bite which he had received, as well as to the

[1] Recherches sur la rage; Paris, 1781.

[2] Van Swieten, Commentaria in Boerhaave aphorismos, § 1138, Rabies canina; Paris, 1758, t. iii. p. 550, 1771.

[3] Id. ibid. p. 556.

nature of his complaint, which he apparently suspected. It is a very remarkable circumstance, which I am anxious to bring forward prominently, that rabid individuals persistently conceal the probable cause of their complaint. It would seem as if they recoiled from the fearful truth, and dreaded to acknowledge it to themselves or to others. In the present instance, a canteen-woman informed Dr. Peter of the bite. Death occurred within thirty-six hours. Another peculiarity of the case consisted in the most exquisite exaltation of some of the senses; the scent of some lilacs in flower distressed him, although they were at a distance of about thirty metres from him: the least movement of the air, as by the opening of a door, made him complain of a disagreeable sensation as if his face were slapped, and caused him to jump in bed.

Nymphomania has been sometimes noted in women, and in the case of a young woman who died of hydrophobia in the year 1861, this symptom was mentioned by Dr. Bricheteau, who published the case. The fact has been only noted during the period of excitation of rabies; yet Mr. B., even while he was given to sadness and melancholy, had shown sexual aptitudes which had created much surprise, as for a long time he had been very frigid.

You will find many cases of hydrophobia recorded in books which treat of this complaint, but I will nevertheless relate to you some of the cases which came under my own observation.

In 1823, when I was a pupil of Bretonneau's, the son of a joiner, seven years old, was brought to the Tours hospital. Bretonneau at once recognised hydrophobia on seeing the boy. The poor little fellow could not lie quietly in his bed, and was singularly agitated; all bright objects frightened him, and the unfolding of a napkin before him brought on a paroxysm; in fact, every thing alarmed him. He died three or four hours after his admission into the hospital. He had been bitten by a mad dog three months previously.

This was the first time that I had ever seen a case of hydrophobia, and the impression, therefore, which it made on me, was a lasting one.

A few years later, while I was a resident assistant in the Charenton hospital, Mr. Calmeil and I were going round with Esquirol, when I was summoned to see a patient who had just come to the infirmary, and who, it was said, had a strange aspect. He was about twenty years old: everything alarmed him, and his face expressed terror. I remembered the little boy I had seen in the Tours hospital, and going back to Esquirol, I informed him that a man suffering from hydrophobia had just been admitted into one of his wards. The man could not swallow, and the sight of bright objects created

a strange terror in him. His intellect was perfect, and he told us himself that he had been bitten in the leg by a dog five months previously, but had not felt any uneasiness on that score. Esquirol ordered him to be put into a cell: the poor fellow apologised for the trouble he gave, saying that he had been unwell for the last two days only, and that he had not slept during the previous night. By Esquirol's orders, he was tied to his bed without his offering any resistance, but when he was asked to drink, he was seized with fearful spasms and convulsions as soon as the glass was raised to his lips. His pulse was full, and he was greatly agitated. This was a time when Broussais' doctrines had many followers; so that the patient was ordered to be bled. I held his arm, and as he kept spitting all the time, my face was covered with his saliva. On a napkin being thrown over his head, he took fright, and became seized with convulsions; the blood ceased to flow, and he died. Had the bleeding anything to do with the rapidity with which death occurred, or did the patient die in consequence of a spasm of the respiratory muscles, such as is most commonly observed in individuals who have reached the second stage of the hydrophobic disease?

In 1831, I saw with Bonnet (of Lyons) in one of Mr. Récamier's wards at the Hôtel-Dieu, a man still young, from whose aspect, expression of the face, and extreme agitation we diagnosed hydrophobia. He had been bitten, seven or eight months previously, by a cat which had disappeared from the house and had not since returned. Messrs. Magendie, Caillard, Petit, and Récamier entertained no doubt as to the nature of the case. The man could only drink with very great difficulty; he was constantly spitting, and was very much agitated. By Magendie's advice, a mixture containing 36 drops of the officinal preparation of prussic acid was ordered, but no sooner had he taken this than he looked as if he had been struck by lightning, his pupils were dilated and immovable, and I thought he was dead. I left Bonnet with him, and ran to the dispensary to ascertain whether some mistake had not been made in preparing the mixture. On then hearing that the strong instead of the dilute acid had been used, I left the hospital to go to the *Bureau Central*, fully persuaded that the patient had been poisoned. Soon afterwards, Bonnet came and told me that the man still lived and consented to drink; that his pupils were still dilated, but that he was no longer agitated. Now it was a question whether the prussic acid had done him good? I saw him an hour afterwards; he was again very agitated and was unable to drink. I then prescribed six drops of dilute prussic acid; there was no error this time, and the dose of the drug was six times less, yet the patient had no

sooner attempted to swallow the mixture than he was as rapidly thrown into the same condition as before. By degrees, however, breathing was restored. After this, we felt little inclined to ascribe to the prussic acid the phenomena we had witnessed, and we regarded them as the consequences of the efforts of swallowing which, in such cases, bring on spasms of the respiratory muscles and rapid asphyxia. A third attempt was made to get the patient to swallow two drops of prussic acid in a mixture, but he became agitated again, and the convulsive paroxysms growing more and more frequent, death supervened forty-eight hours after the invasion of the complaint.

These cases are not given with full details, it is true; but it was a well-ascertained fact that all these patients had been bitten, and that after a period of incubation of varying duration, and without any appreciable determining cause, mental or physical, they had felt general discomfort and been very much agitated. Inability to swallow fluids had soon come on; the sight of fluids or of bright objects had been enough to bring on convulsions, at first clonic and then tonic, and lastly, the patients died of asphyxia through spasm of the respiratory muscles. It cannot be affirmed that death always takes place by asphyxia, because the practitioner is not always there to witness the mode in which the fatal termination occurs; but there is such marked asphyxia during the paroxysms, and dissection so often discloses after death the lesions proper to that condition, that one is authorised to believe that most commonly the patient dies of asphyxia during a paroxysm. I shall, by and by, analyse what takes place during a paroxysm in hydrophobia, and I shall enquire at what time the patient is in danger of dying.

The following case, which was communicated to me by Dr. Eugène Fournier, will show you the part played by asphyxia as a cause of death in hydrophobia. On June 18, 1860, a man aged 27, a joiner by trade, was brought in the evening to Beaujon Hospital, by order of the police commissary of Batignolles, because he was thought to be suffering from rabies. He had been bitten, two months previously, by a small dog which he was teasing. The dog had disappeared, and never returned. The bite had inflicted a small wound in the ring finger of the right hand which healed up very rapidly; it was not cauterized, and no trace of it could be found. The man went on with his occupation for two months, without experiencing any peculiar sensation. But on June 15, he felt some discomfort and had nausea. On the following day, whilst working in a room, he nearly fell down from a chair on which he had got up, and on trying to save himself by catching hold of a piece of furniture with his right hand, he felt an acute

pain in his right arm. As this pain persisted, he told people that hydrophobia was seizing him in the arm where he had been bitten by a dog suspected to be mad.

He felt ill on June 17, and remained in-doors. The next morning, early, somebody went and told his sister that he was delirious. Still he recognised his sister and stoutly refused to be taken to the hospital, so that a police warrant had to be obtained before he could be removed, and it was only with very great difficulty that he was brought to Beaujon Hospital. He got pretty calm when he was once in bed, and quietly said that he had felt an inclination to be sick and had had an indigestion, but could not conceive why violence was had recourse to to bring him to the hospital, where, he added, he was very glad to be. When he was questioned as to the origin of his complaint, he turned his eyes away, and refused to admit that he had been bitten by a mad dog. His skin was hot, especially about the face, his pulse frequent, but not tense; the pupils were neither dilated nor contracted, the eyes were sunken in and surrounded by a bluish circle. He had a headache, and no appetite, and did not complain of thirst. He was offered drink, but refused. He was left by himself in a room, confined to the bed by a strait-jacket. Shortly afterwards, however, fearful cries were heard, and on hurrying to him, he was found in convulsions: his face was livid, and asphyxia seemed to be imminent. He spat at times, but there was no foam about his mouth. Other paroxysms probably came on, and death occurred at half-past eleven, about three hours after his admission into the hospital. No treatment had been attempted. There was great cadaveric rigidity three hours after death, and there was noted subcutaneous emphysema of the anterior aspect of the throat.

The body was examined about thirty-six hours after death. There was congestion of the meninges and of the cerebral substance. The lungs were crepitant, bluish on the surface, black on section, and gorged with blood. There was a perforation in the upper lobe of the right lung, near which there was sub-pleural emphysema, while air bubbled up through it when the lung was compressed. The congestion of the lungs, and the presence of emphysema, at first sub-pleural and afterwards cervical as well, from rupture of the lung-tissue, prove that during the last moments of life there must have been extreme obstruction to breathing from a spasm of the glottis, which by shutting up the natural aperture of exit of the air compressed inside the lungs during the convulsive paroxysm, led to rupture of the lung-substance, and consequently to emphysema.

Most authors entertain no doubt as to asphyxia being the cause of death in hydrophobia, and in a case related with very

great care and commented on with rare talent by my colleague in the hospitals, Dr. Bergeron, the details given show that the patient died of asphyxia. In this instance, however, the asphyxia seemed to have been *gradual*, not *sudden*; for it is stated in the report of the case[1] that lividity of the face began to show itself about three hours before death, and became more intense after a time, and that speech was impeded by an accumulation of bronchial mucus in the fauces; and, lastly, that during the half hour which preceded death the face was purplish and bedewed with perspiration. On examination of the body there were found, in this case also, marked signs of asphyxia; the whole venous system was gorged with blood, the meninges and the brain-substance were markedly hyperæmic, and the lungs crepitated at the apex and in front, but were hard and less crepitant posteriorly, while their colour was of a deep brownish-red tint. When incised, a good quantity of black blood exuded from them. In the posterior margin of the right lung were a few small apoplectic centres, and especially blood-imbibition.

Now, gentlemen, can it be said that individuals affected with hydrophobia must all necessarily die of asphyxia? I would not dare to state this positively, because of the rapidity, the suddenness with which death occurs in some cases. Yet, I believe that rapid asphyxia from closure of the glottis, or slow asphyxia from repeated spasm of the respiratory muscles, is the most frequent mode of termination of this complaint. The clinical reports and the anatomical details to which I have called your attention seem to me to leave no doubt on this point.

Before I describe the chief symptoms of hydrophobia proper, I wish to relate to you a few cases of mental hydrophobia, a special form of the complaint, which is brought on by emotion on seeing rabid individuals, or on hearing a description of real cases of hydrophobia.

In the spring of the year 1828, I was engaged with my colleague at the Academy, M. Leblanc, and with Dr. Ramon, in investigating the rot-disease which was rife among the sheep of the Sologne. He had just inoculated with the disease three hundred sheep belonging to M. Joupitre, mayor of the department. Whilst talking of virulent diseases in general, this gentleman told us that he had been affected with hydrophobia. A farm-dog had tried to bite his arm, and about the same time had bitten a good many beasts which died of rabies. A few months afterwards, on Easter Sunday, after service, and at a breakfast at which every one had done his best to make up for the rigid abstinence of the past Lent, M. Joupitre exclaimed suddenly that he was seized with hydrophobia. He could not eat or

[1] Archives de Médecine, 1862.

drink any more, and our host was already beginning to rave, when his wife, who only believed that he had eaten too much, persuaded him to tickle his throat with his fingers. The advice was good, for copious sickness was brought on by the manœuvre, and nothing more was said about rabies.

That same year I happened to relate M. Joupitre's case to a presiding judge in chambers, who, in his turn, told me that he also had once believed himself to be seized with hydrophobia. He used to go out riding frequently, and a sporting dog which generally accompanied him, often jumped to kiss the hand with which he held his whip. During one of these rides they once met a flock of sheep, after which the dog ran, biting those he could catch. The animal still heard and obeyed his call, but he had a strange aspect. Again he ran after and bit dogs, cows and oxen, and lastly swam across a river; a few hours later, he died. A short time after this, the judge heard that many of the beasts that had been bitten by his dog, had died of rabies. This news alarmed him, because he recalled to mind that on the same day the dog had licked his right hand several times. On examining his hand, he found several small scars on it, and seized with terror upon this, he no longer dared touch water to shave himself, and fully believed he had hydrophobia. A medical man, who was sent for from Orleans, tried in vain to calm his fears: for several days he was excited and delirious. At last, being told over and over again that persons seized with rabies died very rapidly, and that he could not therefore be rabid since his dread of water dated already ten days back, and after reading in books about the duration of confirmed hydrophobia, he allowed himself to be persuaded, and his dread of water vanished as soon as he became convinced that he should have died long ago if he had been rabid.

You see then, gentlemen, that a nervous kind of hydrophobia may develope itself under the influence of intense mental emotion, or when certain excesses or special conditions induce dysphagia or a disgust for food; and medical men may themselves be deceived, if they do not bear in mind the length of the period of incubation of real hydrophobia, and the course of this fearful malady, which invariably kills in the space of three or four days from the first manifestation of the symptoms. It is important that one should be aware of this cause of error, which might have fatal results, because I have known medical men—men of strong minds and of courage, who although well aware of the conditions needed for the development of rabies, for several months, and even years, after having attended persons suffering from hydrophobia and dissected their bodies, were seized with more or less continued dysphagia on the mere thought and recollection of the awful scene which they had witnessed. Time

alone got rid at last of this nervous susceptibility, which manifested itself in the shape of spasm of the pharynx, and they cured themselves of it by appealing to their knowledge of the disease, and by forcing themselves to drink some liquid whenever they felt the sensation coming on.

I have related to you, gentlemen, several cases of true rabies occurring in man, and I might easily swell their number by telling you of cases of young children bitten by mad dogs, who came under my observation. I have also given you instances of nervous hydrophobia, so that we might now review together the chief symptoms of human rabies; but before doing so, I wish to give you a sketch of canine rabies. My intention is not merely to draw a parallel between rabies occurring in man and rabies developed in dogs, but I am anxious also to teach you how to recognise canine madness, and thus furnish you with the best preservative against human rabies. For if hydrophobia could be always recognised in a dog, the creature would be immediately sacrificed, and the chances of inoculating the disease to human beings would be thus lessened.

In one of the late discussions on rabies at the Academy of Medicine, M. H. Bouley, clinical professor to the school at Alfort, drew a striking picture of rabies canina, based on what he had seen himself and on quotations from Youatt's work.[1] There are three well-marked stages of the complaint in the dog. The first is characterised by melancholy, depression, sullenness and fidgetiness; the second by excitement, by rabid fury; and the third and last, by general muscular debility and actual paralysis.

Whether the disease originated *de novo*, or was communicated, the dog looks ill and sullen after a period of incubation of very variable length: he is constantly agitated, turning round and round inside his kennel, or roaming about if he is at large. His eyes when turned on his master or friends of the house have a strange look in them, expressive of sadness as well as of distrust. His attitude is suspicious, and indicates that he is not well; by wandering about the house and the yard, he *seems to be seeking* for a remedy to his complaint. He is not to be trusted even then, because although he may still obey you, yet he does it somewhat slowly, and if you chastise him, he may, *in spite of himself*, inflict a fatal bite. In most cases, however, a mad dog respects and spares the persons to whom he is attached. But his agitation increases; if he is in a room at the time, he

[1] Bulletin de l'Académie Impériale de Médecine, Paris, 1863, t. xxviii. p. 743, *et seq.* See also, Rapport général fait à la demande du gouvernement sur divers remèdes proposés pour prévenir ou pour combattre la rage, by Bouchardat. Bulletin de l'Acad. Imp. de Méd., Paris, 1852, t. xviii. p. 6 to 30, and 1855, t. xx. p. 714 to 727.

runs about, looking under the furniture, tearing the curtains and carpets, sometimes flying at the walls as if he wished to seize a prey. At other times, he jumps up with open jaws as if trying to catch flies on the wing; the next moment, he stops, stretches his neck and seems to listen to a distant noise. He probably has then hallucinations of sight and hearing, seeing objects that do not exist and hearing sounds which are not emitted. This delirium may still be suddenly dispelled by his master's voice, and, according to Youatt, 'dispersed by the magical influence of his master's voice, all these dreadful objects vanish, and the creature creeps to his master with the expression of attachment peculiar to him. There follows then an interval of calm; he slowly closes his eyes, hangs down his head, his fore-legs seem to give way beneath him, and he looks on the point of dropping. Suddenly, however, he gets up again; fresh phantoms rise before him; he looks around him with a savage expression, and rushes, as far as his chain allows him, against an enemy which only exists in his imagination.' By this time already, the animal's bark is hoarse and muffled. Loud at first, it gradually fails in force and intensity, and becomes weaker and weaker, apparently indicating incomplete paralysis of the muscles of the jaws, just as the dropping down pointed to paralysis of the muscles of his fore-legs. In some cases, the power of barking is completely lost; the dog is dumb, and his tongue hangs out through his half-opened jaws from which dribbles a frothy saliva. Sometimes his mouth is perfectly dry, and he cannot swallow, although in the majority of cases he can still eat and drink. When he has vainly attempted to swallow, he probably believes that it is because some foreign body sticks in his throat, for he puts his muzzle between his paws and works with them as if he wanted to get rid of this. Although he can no longer drink, people are misled into the belief that he does so from his lapping fluids with great rapidity. On close examination, however, the fluid is found to keep the same level in the vase which contains it, and one can see that the dog does not in reality swallow, that he does not drink, but merely bites the water. Although he cannot swallow fluids, he can still, in some cases, swallow solids, and he may then swallow anything within his reach, bits of wood, pieces of earth, the straw in his kennel, &c. This circumstance is one of very great importance to bear in mind, because when the body of a mad dog is dissected, a good many substances which have not been digested may be found in his stomach, and do thus furnish a proof of his complaint.

One period of the disease does not pass suddenly into another, but by an easy transition. Even in the first stage, that of depression and melancholy, the animal is from time to time

very agitated, and shifts his posture. This agitation increases to a considerable degree, and in the second stage constitutes the rabid fury which characterises this period, together with the hallucinations of sight and hearing. During this second period the animal drops down in a state of exhaustion, after paroxysms of rage; he seems completely prostrate, his head hangs down, his limbs give way under him, and he can no longer swallow. These are signs of incipient paralysis.

Towards the close of the second stage, the dog often breaks his chain, and runs far away from his master's house; he wanders about in the fields, seized from time to time with paroxysms of fury, and then he stops, from fatigue, as it were, and remains several hours in a somnolent state. He has no longer the strength to run after other creatures, although if he be worried, he can still gather strength to fly at and bite an individual. If he be not destroyed, as he wanders about, he generally dies in a ditch or in some retired corner. He apparently perishes from hunger and thirst and intense fatigue; but veterinary surgeons do not say that he dies from asphyxia brought on by spasm of the pectoral muscles or by convulsions.

Such, gentlemen, are the chief symptoms of rabies when occurring in dogs. It has not been my intention to give you a complete description of rabies in the lower animals, but merely to teach you how to recognise it in the dog. Van Swieten had already divided into three stages the course of confirmed hydrophobia in the human subject, the prominent characters of which were melancholy, furor, and asphyxia. When we enquire into the course of the symptoms of rabies in man, we shall see that this clinical division is founded in fact. This terrible malady is always inoculated to the human subject. It may have an incubation-stage, varying from a few days to a year; but the disease generally shows itself from one to three months after the infliction of the bite. The cases are rare in which it developed itself after three months, and still more rare in which it came on from the sixth to the twelfth month, and one is almost authorised, from the statistical observations that have been made, to question the authenticity of the cases in which the disease set in a year after the person had been bitten. *A fortiori*, must one regard with suspicion those instances of the disease in which the incubation is stated to have been more prolonged. These latter may not have been cases of true rabies, but of nervous hydrophobia, similar to those which I related to you, and in which the mere recollection of this awfu complaint sufficed to bring on a more or less prolonged dysphagia.

During the incubation-stage there is no disturbance of health, no symptom which may excite suspicion, and, according to Van Swieten, persons who afterwards die of hydrophobia may

contract diseases of various kinds, and even virulent diseases, such as variola, without the course of the rabies being thereby modified in the least. The virus of variola has, therefore, no influence on that of rabies, since it does not retard the evolution of the latter complaint, if it shows itself during the incubation stage.

After the incubation stage has lasted two or three months, the person who has been bitten suddenly becomes unusually sad; he either does not suspect his complaint, or, if he remembers having been bitten, carefully avoids to mention the circumstance, and seeks amusement away from home. But wherever he may be, his sleep is disturbed, and he often starts up; he feels constantly fidgety, sighs deeply, shuns his friends, seeks solitude, and begs that perfect silence be observed about him; any attentions shown him increase his restlessness and agitation.

Aggravation of these symptoms indicates the beginning of the second stage of the disease. Other phenomena show themselves also. The patient complains of a sense of discomfort about the præcordial region; his respiration is sighing, his pulse irregular, as was noted in M. B. and in several of Van Swieten's cases. These disturbances of respiration and circulation, and the sadness and agitation, point to an already marked modification of the nervous system. This goes on increasing, and rigors supervene, which are true convulsions of all the muscles of the body. Then a symptom shows itself, which is nearly constant in confirmed rabies attacking the human subject, namely, *a dread of water*.

The sight of water is frequently sufficient to bring on shuddering; but it is when the patient carries water up to his lips that he is seized with the terrors characteristic of the disease, and with those convulsions of the face and of the whole body which make so deep an impression on the bystanders. A rabid individual is perfectly rational: he is thirsty, tries to drink, and commands his hand to raise to his lips a glass of water; but the liquid has no sooner touched his lips, than he draws back in terror, and sometimes exclaims that he cannot drink; his face expresses pain, his eyes are fixed, and his features contracted; his limbs shake, and his body shivers. The paroxysm lasts a few seconds, and then quiet seems to be gradually restored; but the least touch, nay, mere vibration of the air, is enough to bring on a fresh paroxysm, so great is the cutaneous hyperæsthesia in some cases. The patient cannot wash his hands or face, or comb his hair, without being at once threatened with convulsions.

During the intervals of calm, he sometimes complains of pain in the stomach and of nausea; when he is actually sick,

he brings up greenish, porraceous matters.—I have already mentioned priapism. This peculiar condition of the genital organs is exceedingly painful, and patients usually express their sufferings in terms which cannot be repeated here. In some cases the patient is seized with sudden terror; he turns abruptly round, fancying that somebody calls to him; there are hallucinations of sight and of hearing. Dr. Bergeron's patient heard the ringing of bells, and saw mice run about on his bed.

You must have been struck, gentlemen, with the many points of resemblance between these two first stages of human rabies and canine madness. In the human subject and in the dog the same symptoms show themselves: melancholy, sadness at the onset of the complaint, a desire to go away from home and to shun friends, agitation, restlessness, and hallucinations.

But as the agitation and the sadness increase, the second stage begins. In man, however, the disorders of innervation may differ from those in the dog, although satyriasis and hallucinations may be present, and the nervous system be deeply affected in both. In the human subject there is cutaneous hyperæsthesia; in the dog, on the contrary, sensibility seems to be abolished, for rabid dogs have been known to seize between their teeth a red-hot poker, without evincing any sign of pain, and they scarcely move away when fire is set to the straw or the tow on which they may be lying. Lastly, whereas a rabid man shows a dread of water, a mad dog seeks water, and jumps into rivers, and bites the water; but he cannot drink, because he cannot swallow, and as in man, the dysphagia is probably dependent on spasm of the pharynx. He has hallucinations also, for he flies at imaginary objects, and hears imaginary noises. In the human subject there are at first clonic, and then tonic convulsions of the muscles of the life of relation, of those of deglutition, and of respiration, during a paroxysm; his aspect strikes one with fear, but he has no desire to bite, and does not strike the persons about him. A mad dog, on the contrary, flies at all the beasts which he meets with, and especially at dogs. In his rage he bites, while a horse kicks and tears with his teeth, and a ram or bull butts with his head. A dog bites because his teeth are his weapons of defence and offence; but we may well wonder why a rabid man does not strike with his arms. The reason is that during a paroxysm of rabies, a man is not in a state of furious anger, but is merely convulsed.

In the third stage of human and canine rabies, there are many points of resemblance, but as marked differences also. In both cases, each period of the disease is not abruptly separated from the others; but, as Van Swieten pointed out, the symptoms

become more serious, and as fresh ones show themselves, there are degrees, rather than stages, of the disease.

In the third and last stage of the complaint, there is more intense thirst, while there is greater incapacity for drinking: the voice becomes hoarse, at first intermittingly and then continuously, probably in consequence of spasm or paralysis of the laryngeal muscles. In the last hours preceding dissolution the patient's mouth is often full of a whitish froth, which he spits out constantly. Now, is this froth merely the result of the constant agitation of the saliva through the movements of the cheeks, the lips, and the tongue; or is it due to the patient's inability to swallow his saliva; or, again, does it consist of a mixture of saliva with a variable amount of bronchial froth, driven into the mouth by spasm of the pharynx?

The sight of this froth, and the constant spitting, sometimes alarm the patient himself: he thinks that the matter which he expectorates may do harm to the persons about him, and, as in the case reported by Dr. Bergeron, he begs that nobody is to come near him, and dreading for others the contact of his lips, he refuses to kiss his friends, he dreads to communicate his complaint to them, *aliis à se metuens*, as Boerhaave has it. Van Swieten mentions that a man communicated rabies to his two sons by kissing them, although he does not wish to draw absolute conclusions from the case. Medical men of the present day, however, are little inclined to believe that any danger can accrue from the contact of the saliva of a rabid individual with unbroken skin; but even those who think that there is then no cause for fear, take very good care to wash thoroughly the parts which may have been in contact with the saliva of a rabid individual. I am of opinion that one should prudently avoid all contact with the patient's saliva, as the dog's saliva is capable of imparting the disease to man, and as it has been shown by experiments made by Magendie and Breschet in 1813, and afterwards confirmed by Renault, at Alfort, that dogs have become mad after being inoculated with the saliva of a rabid man.

As the voice becomes hoarse, and the patient keeps constantly spitting, the convulsive seizures get more and more frequent, and recur spontaneously, without any determining cause. The close of each seizure is attended with spasm of the respiratory muscles, and signs indicating some obstruction to the breathing. On this spasm lasting a long time in one of the seizures, the patient dies asphyxiated, *mors convulsiva cum summâ in respirando angustiâ*. Rabies, in the dog, often lasts several days; in the human subject death always occurs within four days after the first rigor and difficulty of deglutition have set in. The mad dog apparently dies palsied, while the human patient dies from

a tonic convulsion of the respiratory muscles. This is an important difference which should be noted in the final stage of canine and human rabies. Van Swieten, however, in his comments on Boerhaave's 1138th aphorism, relates a case of hydrophobia occurring in man, in which death was not preceded by convulsion, or even by struggles, and seemed to result from general paralysis, *ac si universalis paralysis mortem induxisset.*

There is no symptom during the period of incubation of the disease which indicates that the individual has been inoculated with the virus of rabies. In a great many cases, no ill effects have followed a bite inflicted by a rabid dog. We may then suppose that no virus was deposited in the wound; or, if the view be not admitted that some individuals are not susceptible of the poison, it may be conjectured that from some special conditions, no absorption of the poison took place. Whatever hypotheses may be started on this point, facts prove that of several persons and several animals bitten by the same mad dog, a few only become rabid. During the period of incubation, which may extend over several months, no sign will indicate which individuals out of the number will fall victims to the bite. No change in their habits or their functions awakens suspicion; and yet I wish you to remember that one of my patients, some time before the invasion of the complaint, exhibited a return of sexual aptitude which he seemed to have lost a long time ago. If, during the period of incubation, doubts and fears may exist, all uncertainty comes to an end when the stage of invasion begins. The muscular debility complained of in many cases, the restless sleep out of which the patient starts up, his continual fidgetiness, his suspicious breathing, his sadness and search after pleasure, and then his love of solitude, must awaken terrible fears in the practitioner, especially if there be no moral causes or no organic lesions to account for these symptoms satisfactorily. The intense thirst, general muscular pains, and rigor which might at first be ascribed to some grave febrile affection, are followed by a symptom which is almost pathognomonic of rabies, namely, a sudden difficulty in swallowing liquids, water in particular. When there is complete inability to drink, and when this dysphagia is immediately succeeded by tremor on the patient carrying some liquid to his lips, all illusion is dispelled, and it becomes clear that the patient is under the fatal influence of the virus of rabies. There is such a thing as nervous hydrophobia, true dysphagia brought on by a dread of rabies, and I have related to you examples of it; but the sudden invasion of this complaint, generally coming on through the person recalling to mind or hearing the relation of a case of true hydrophobia, and the duration of the dysphagia over the period of four days, are amply sufficient to characterise the

complaint, and to enable the practitioner to persuade the patient that he is suffering from mere nervous symptoms which will vanish as soon as he ceases to fear. Besides, in nervous hydrophobia, there is dysphagia only, but no general convulsions, the spasm affecting the pharynx alone, while the breathing goes on with regularity.

Maniacs sometimes also evince a dread of liquids, and refuse to drink, and like persons suffering from rabies in the second stage, they are exceedingly agitated and loquacious, and have hallucinations, but they never have general rigors and spasmodic convulsions. They are, besides, delirious on all subjects, whilst a rabid individual retains all his reason, although he may occasionally have transient hallucinations. He is anxious to get well, and believing that his complaint is merely due to his inability to drink, he submits to any treatment; and once the paroxysm of excitement over, he allows a strait-jacket to be put on him without offering the least resistance. A maniac has lost his reason; in rabies, on the contrary, as Boerhaave stated long ago, even in the last stage of the disease, the patient retains his firmness and common sense, and requests the persons about him to keep some distance from him, because he dreads lest he should communicate his complaint to them.

I may here mention, that in the beginning of this century, a Russian physician, Dr. Marochetti, in a memoir on hydrophobia, and Dr. Xanthos, of Siphnos, in a letter to Hufeland, called attention to the presence, on the under surface of the tongue, near the frænum, of pustules or vesicles of a special character, during the stage of incubation of rabies. These had been long known in Greece under the name of *lyssi*. Drs. Marochetti and Xanthos did not claim for themselves the credit of this important discovery: it was traditionally known, they said, in Russia and in Greece; and they had been told that if these vesicles or pustules were laid open in time and cauterized, all manifestations of rabies were prevented. Dr. Marochetti, in particular, frequently put this plan to the test, and succeeded completely; he recommends at the same time, it is true, the use of a ptisan of *genista-tinctoria* (Linnæus) which has for a long time been used in Ukraine against rabies. The presence of an eruption under the tongue subsequent to inoculation with the virus of rabies, seems to me to be a fact of such importance, that it is my duty to call your attention to it, as it has been observed by men whose scientific honesty we have no right to suspect. I must observe also, that if the presence of this vesicular eruption has not been ascertained in France since the publication of Dr. Magistel's work[1], it is because practitioners have not, in

[1] Mémoire sur l'hydrophobie, or Journal de l'hôpital de Burley, Paris, 1824.

general, looked for it during the period of incubation of rabies, although Drs. Marochetti and Xanthos have particularly stated that it showed itself during the first few days after the inoculation. Dr. Magistel says, in his memoir, that he saw the *lyssi* in different individuals on the sixth, the eleventh, and the twentieth day, and that, after the twenty-second, he never succeeded in finding them, although he looked for them until the thirty-fourth day after the inoculation of the virus. It would seem, therefore, that this peculiar sublingual eruption may be met with in a certain number of cases of rabies, but that one must know how, and particularly when, to look for it, namely, at the commencement of the incubation-stage, and not in the period of invasion or of confirmed rabies, since the eruption has disappeared by that time, without leaving any trace behind. In the present state of science, we may not perhaps have the right to deny, as has been done, the presence of lyssi in rabies. This eruption should, therefore, be searched for in persons who have been recently bitten by a rabid animal, and whose wounds have not been cauterized.

The accuracy of the statements made by Drs. Marochetti and Xanthos may easily be tested, because the wounds inflicted by rabid animals are rarely cauterized sufficiently early and to a sufficient depth so as to prevent absorption of the virus, so that in a certain proportion of cases, the presence of lyssi ought to be made out from the third to the twentieth day after the inoculation.

I need not dwell on the advantages that would be obtained if the statements made by Drs. Marochetti, Xanthos, and Magistel were confirmed. Rabies could then be diagnosed during its incubation stage, and if by laying open and cauterizing these vesicles the ulterior manifestations of the disease can be prevented, the complaint could be cured, as soon as the sub-lingual eruption was detected.

I cannot, therefore, too strongly recommend practitioners to look out for the presence of lyssi in individuals who have been exposed to the risk of being inoculated with rabies. The examination should be made regularly twice a day, according to Marochetti, because the eruption does not show itself on a fixed day, and the vesicles break easily. The same authority adds, that the eruption comes out at an earlier period in proportion to the amount of poison deposited in the wound, and that the invasion of the confirmed disease comes on also earlier, according to the early date of the appearance of the eruption.

There seems to have been of late a disinclination to attach any importance to the presence of this eruption, and it has been said that it was a very extraordinary circumstance, without its analogue in pathology, that the virus of rabies should be

localised—stored up in a particular region. I cannot entirely concur in this opinion; and without desiring to prove that this localisation of the virus is a perfectly natural phenomenon, which might have been foreseen, I will merely call attention to the fact that in most virulent diseases, a primary localisation of the virus may be detected, in a particular tissue organ, and that the disease gives rise to general manifestations only secondarily. Thus, in eruptive fevers, we see the morbid principle affect the skin primarily, and the lumbar portion of the spinal cord in small-pox, the bronchial and laryngeal mucous membrane in measles, and the kidneys in scarlatina. We see syphilis limit itself in the first instance to the lymphatic ganglia in the groin and in the occipital region, and station itself for a time in the lymphatic system before giving rise to secondary manifestations in mucous membranes and in the skin. Again, we see the poison of glanders affect the mucous membrane of the nose in the beginning, and it is only after a time that other mucous membranes are involved, together with the skin, the cellular tissue, the joints and viscera.

If in virulent diseases in general, therefore, the virulent principle has a primary elective seat, why should one refuse to admit, on *à priori* grounds, that the virus of rabies, when inoculated into any part of the body, can act primarily on a determinate and localised region? Why should any surprise be felt at its selecting the free extremities of the excretory ducts of the salivary glands, when no doubt is now-a-days entertained as to the saliva itself being the vehicle of the poison? Again, why wonder that a special eruption should be confined to the region where the excretory ducts of the sub-lingual and sub-maxillary glands terminate, when experiments by Claude Bernard have proved that all the salivary glands do not possess similar properties? Lastly, when it has been experimentally shown that certain substances, such as iodine and the iodides of potassium and of iron, are more rapidly eliminated by the salivary glands than by any other organs, why should it not be admitted that these glands may eliminate an organic poison, a virulent principle which, after a definite time and by virtue of special circumstances, lodges and is stored up in the extremities of the excretory ducts of these glands, or in the salivary follicles in their vicinity?

Marochetti thought that the virus which had been thus deposited was after a certain time absorbed, and then gave rise to all the symptoms of confirmed rabies. He, therefore, followed the traditional practice of the inhabitants of Thessaly and Ukraine, and made an early incision through the vesicles, so as to give issue to the virulent matter, and then cauterized them with a red-hot iron. He affirms that this plan always

succeeded in the numerous cases which came under his observation in Ukraine. One cannot take too much pains to look for this peculiar eruption, since it would seem to be the only sign by which the incubation-stage of rabies can be diagnosed, and since by cauterizing it, one may arrest the fatal progress of the disease.

In a great many old books, you will find that the first symptoms announcing the invasion of the disease, show themselves at the seat of the bite. Boerhaave himself states that the wound becomes painful again, and that vague pains subsequently show themselves in the neighbouring regions also. It has been further said by some that wounds which had healed opened afresh; and Salius Diversus, who fancied that he had discovered an infallible sign of threatening rabies, asserted that a peculiar pain was set up at the seat of the bite, and from there ascended by insensible degrees to the brain in the space of 3 or 4 days, and gave rise to vertigo. This pain would be, therefore, a kind of aura, analogous to the aura of epilepsy and hysteria, with this difference, however, that its progress upwards is very slow.

When men like Boerhaave and Van Swieten declare that such phenomena have been noted, no one can refuse to believe that they may occur, although it should be observed that in cases recorded in modern times by careful practitioners, no allusion is made to any such phenomena. Thus, in Dr. Bergeron's case, which is given with such full details, it is distinctly stated that the scar left by the bite *had undergone no change* and was not painful. In the case under my care, in St. Agnes ward, no painful scar could be found on the hand that had been bitten. Lastly, in the cases reported by Dr. Peter and by Dr. Eug. Fournier, not the slightest trace of the bite could be seen; yet, I must recall to your memory that two days before he was brought to the Beaujon Hospital, the subject of Dr. Fournier's case complained of pain in his right arm, and said that he was being probably *seized with rabies in the arm* which had been bitten.

The *prognosis* of rabies, in the stage of invasion, when dysphagia and convulsions have shown themselves, is always unfavourable. Nothing that has been tried has ever succeeded in arresting the fatal progress of this fearful complaint, and death occurs more rapidly in proportion to the frequency of the convulsions.

I now pass on to the *etiology* of rabies in the human subject.

The last discussion which took place at the Academy on Rabies, and in which Messrs. Vernois, Bouley, and Tardieu joined so brilliantly, referred chiefly to the etiology of the

disease in the dog and the human subject. From the facts stated in the course of that discussion and the results of statistical investigations, an important inference can be drawn, namely, that rabies very rarely occurs in the human subject. In France, with a population of more than 36,000,000, there are only every year, on an average, from 20 to 25 cases of rabies, that is, less than two cases to a million of people. And yet there is a certain proportion of bites inflicted every year by rabid dogs, and statistical researches have shown that the bite proves fatal fifty times out of a hundred. These facts would seem to indicate that certain individuals are not susceptible of the influence of the virus. But if we bear in mind that viruses when inoculated take effect in nearly every case, it would perhaps be more rational to believe that every bite does not inoculate the virus of rabies, either from the virus not having been secreted by the salivary glands at the time, or from its being wiped off from the tooth in its passage through the clothes. This last hypothesis is supported by the fact that the most dangerous bites are those inflicted on exposed parts of the body, such as the face and hands.

Rabies is communicated to man by different animals in the following order with regard to frequency: first comes the dog, next the cat, and then the wolf and the fox; in very exceptional instances it has been transmitted by cows and by horses. The chief cause of the difference in the results following the bites of these animals lies in the fact that the first on the list make use of their teeth when they attack man, whilst the last strike with their head or feet. A horse may, however, inflict terrible wounds with his teeth, so that one must be very cautious when he gets near a rabid horse, because his teeth may be charged with virus in a fit of anger.

It is generally said that the period of childhood favours the rapid development of rabies. But this saying seems to me to be one of the results of misinterpreted statistics. If it be true that children are more frequently seized with rabies than adults are, the probability is that this does not depend on a greater morbid susceptibility arising from their age, but on their frequently playing with dogs who bite them when they are rabid, and on their being too inexperienced to recognise the strange aspect of a rabid dog, and too weak to defend themselves or to avoid danger. Can the bite of an angry dog, who is not rabid, give rise to rabies? One cannot conceive how an animal can communicate a virus which he himself has not about him, and if this were unfortunately the case, the number of cases of rabies would be infinitely greater, for there are very few individuals who through life escape being bitten by a dog. Or 'we should be compelled to admit,' as Mr. Bouley remarks,

'that there may exist in the dog a perfectly transient and evanescent rabid condition, during which the animal's saliva is virulent, but after which it becomes normal again.' Such a view would be a pure hypothesis, while facts show, on the contrary, that a dog who has communicated rabies dies himself of the disease. I must not, however, omit to mention a case recorded by Dr. Camille Gros, as having been under Dr. Tardieu's care in the Lariboisière Hospital, and which is one of the rare instances apparently pointing to this as an exceptional cause of rabies.[1]

Van Swieten reports that an old woman died with all the symptoms of rabies, after a wound inflicted on her by an irate cock; but, as he could not admit that a virus not present in an animal could be communicated by that animal, he conjectures that the cock was suffering from rabies which had been imparted to it by a fox. He adds besides, that if rabies could be spontaneously generated in the cock, we ought to be surprised why it does not more frequently occur in England, where this irascible and quarrelsome bird is trained to fight.

Malpighi declares also that his own mother died of rabies a few days after being bitten by an epileptic. But, in spite of the authority of these writers, I believe that the cases which they have recorded are very questionable. We should be equally incredulous as to the cases of spontaneous rabies said to have been observed in the human subject. M. Vernois[2] cited, in proof of the spontaneous origin of rabies in man, cases observed by Dr. E. Gintrac[3] of Bordeaux, and by Dr. Barthez; but as M. Velpeau remarked, and justly in my opinion, there was no absolute proof that there had been no contagion in these cases, for it is not necessary that there should be a bite for rabies to develope itself in man. Some portion of the body, denuded of epidermis, need only be in contact with the virus of rabies; and this may occur on the dog licking a person's hand. Van Swieten also mentions that a young man died of rabies after having bitten his own finger in a fit of passion. It may be that, in this case as in many others, traumatic tetanus was mistaken for rabies.

I myself am of opinion that rabies in the human subject is always the result of inoculation with the virus of rabies, and that those cases in which the disease is said to have been communicated by dogs that were not mad, or to have been generated *de novo*, must be regarded as instances of traumatic tetanus or of nervous hydrophobia.

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[1] Thèses de Paris, 1860. *Considérations sur la rage*, by Camille Gros.

[2] Étude sur la prophylaxie administrative de la rage (Annales d'hygiène publique et de médecine légale. Paris, 1863, t. xix. p. 52).

[3] Journal de médecine de Bordeaux, August, September, and October, 1862.

The pathological changes found after death, in cases of rabies, are only those dependent on the asphyxia which occurs in the last stage. Morgagni has studied with considerable care this part of the subject of rabies in his eighth letter, and the conclusions which he came to are similar to those which have been arrived at from dissections made of late years. Hyperæmia of all the parenchymatous organs is alone met with, as a consequence of the final convulsion.

Dissection gives no clue, therefore, to the nature of the complaint, but an analysis of the symptoms and the etiology of the disease lead one to regard it as a virulent malady. The virus contained in the saliva of rabid animals is the sole source of the contagion of rabies, as has been shown by the experiments made by Professor Renault (of Alfort), and by the circumstances under which rabies affects the human subject after a bite inflicted by a dog, a wolf, or a cat, or after the virulent saliva has been in contact with a denuded portion of the integuments. Rabies is, therefore, a virulent disease which should be placed by the side of glanders, another virulent complaint communicated to man by horses. The virus of rabies remains for a variable time in the system, after its introduction into it, without giving rise to any appreciable lesion, except the sublingual eruption, the presence of which is to be regarded as doubtful until confirmed by other observers. It should be remarked, however, that many authors, previous to Marochetti, had spoken of these small tumours under various names, and as being of very various nature; so that, whatever conclusion may be arrived at bye-and-bye concerning them, it must be admitted that the numerous discussions to which they have given rise seem to tell in favour of their existence.

Ettmuller states that up to the 17th century a good deal of attention had been paid to the presence of these sublingual swellings, and he adds, after quoting statements made by many writers, 'others are of opinion that there is no small worm concealed beneath the tongue of a mad dog, but that the swelling which is found there consists of some of the granular blood which stagnated underneath the tongue, in the ranine veins. I have not come to any conclusion on the point, because I have not sufficient data to go upon.' This great reserve, which Morgagni showed also, should be imitated until further light is thrown upon the subject.

I believe that rabies is never generated *de novo* in the human subject, but is communicated by the dog. We should therefore learn to recognise and guess it even in that animal, as he can so easily transmit it by his caresses or bites. Lastly, after inoculation has occurred, measures should be unhesitatingly employed

which destroy at once all property in the virus, and prevent the fatal evolution of the disease.

Cauterization is, after all, the only measure from which a successful result may be anticipated, and, in order to ensure success, it should be done immediately after inoculation of the virus. Delay allows absorption to take place, and the part should therefore be at once and deeply cauterized. By going beyond the area of the virulent inoculation, a more or less extensive wound will be produced, but which gives rise to no risk, while imperfect cauterization exposes the patient to the risk of dying.

A red-hot iron suffices for destroying the tissues of the bitten part, and it has the advantage of acting quickly, and of leaving behind sloughs which take some time to come away. Récamier recommended the acid nitrate of mercury, because it penetrates the tissues deeply, and rapidly disorganises them. Caustic potash and corrosive sublimate also answer the purpose of destroying the tissues, and rendering them inapt to absorb the virus. Either of these caustics may be used, the first and chief indication being to act quickly and deeply.

Rabies will not be developed if the wound has been cauterized sufficiently early and deeply; but if this has been done inefficiently, is there, we may ask, while the disease is incubating, a symptom which warns us of the threatening peril, and which may be used as a guide to treatment?

The generally accepted opinion is that there is no special symptom to be detected during the incubation-stage, however prolonged it may be. Still, we should bear in mind the facts handed down by tradition, and we should take into account the extreme reserve shown by Ettmuller, who admits the facts, although he adds that he is not in a position to decide on the nature of the tumours which are developed under the tongue of persons inoculated with the virus of rabies; and lastly, we should, until further observations are made on this point, credit the statements made by Xanthos in his letter to Hufeland, and by Dr. Marochetti in his memoir. Besides, we have no cause for doubting the value of the cases recorded by Dr. Magistel. Attention should for the future, therefore, be directed to this point, and I cannot too strongly recommend to you to look for these sublingual swellings in all persons that have been bitten by mad dogs. If such an eruption does really exist, and if by laying open and cauterizing the vesicles the disease can be arrested, our fears will be set at rest.

At the same time that the lyssi are cauterized the treatment recommended by Dioscorides and Celsus should be put in force. Mr. Gosselin has lately advocated the same plan again, which he tried in the case of a girl who had been bitten by a mad dog,

but who did not become rabid.[1] Celsus aimed at renewing the fluids in the body by calling into exaggerated action the skin, the liver, the kidneys, and the intestines, and in facilitating repair by a highly-nutritious diet, open air exercise, and prolonged immersion in water. Mr. Gosselin had probably the same end in view, by inducing abundant perspiration, by repeatedly purging the patient, and by recommending violent muscular exercise, and daily sulphur-baths. This debilitating treatment produced very rapid emaciation, notwithstanding the amount of food taken by the patient. She was discharged well, however, from the hospital, and no symptom of rabies showed itself in her afterwards, although her wound had not been cauterized. We cannot draw any conclusion from a single case of this kind, especially as about one-half only of the individuals bitten by a mad dog become rabid; but as this method is not attended with any serious risks, while it may prove successful, I believe that it should for the future be put in force during the incubation-stage. But if, in spite of all that has been done, rabies should set in, what is the practitioner to do? He may do anything, since the patient is doomed to die. Venesection pushed to syncope has been advocated, with the view of emptying the vascular system, and of getting rid of the virus with the blood at the same time. This treatment has not been attended with good results, however; and when the patient did not die from the prolonged hæmorrhage, he died a few hours afterwards during a spasmodic seizure.

The ancients had a celebrated method of treatment for rabies, called the *sailors' method*. The rabid subject was dipped into the sea, into a river, or a bath simply, until he was nearly asphyxiated. Van Swieten mentions cases in which a cure was obtained by means of this double action of water and asphyxia. Euripides is said to have been cured of rabies by this method, and to have from gratitude written that the sea washed away all the ills of man. But the medical men who sanctioned the sailors' treatment probably meant to act with energy on the nervous system of their patients, for these unfortunates were thrown into the sea when they least expected it, and force was employed when they made any resistance. This is a barbarous plan, which could only be excused if it were always successful. Tulpius had great faith in this treatment, and he affirms that 'in the populous city of Amsterdam, where rabies was common, he never saw a fatal case of this disease when the patient had been dipped into the sea at a proper period.'

This plan was chiefly recommended during the incubation-

[1] Bulletin de l'Académie Impériale de Médecine. Paris, 1863, t. xxix. p. 22.

stage, and at the beginning of the period of invasion. But what is to be done when the mere contact of water with the lips brings on spasms, and do the chief symptoms of this last stage of the complaint, the extreme agitation, the convulsions, the abundant secretion of saliva, furnish us with some indications of treatment? As sleep calms all nervous excitement, and suspends the convulsive paroxysms, it seems very rational to me to treat the symptoms, as we cannot act on the morbid cause itself. Opium in large doses would, by inducing profound sleep, answer the double purpose of quieting the nervous excitement and of delaying the convulsions. Mixtures containing opium are of course out of question, since at this period of the disease the patient cannot swallow; but morphia can be used by the endermic or the hypodermic method. Large doses of this salt can be very rapidly introduced into the blood, either by sprinkling with it the raw surface of a blister raised by strong liquid ammonia, or by injecting it under the skin. When once sleep has been procured in this manner, it should be kept up as long as necessary, that is to say, until the disappearance of all spasm as the patient wakes up.

Chloroform might be also used against the spasms of rabies, for the power of chloroform-inhalations to stop all convulsion is well known; but in order to obtain satisfactory results the convulsions should be forestalled, and with that view the patient should be for several hours every day kept under the influence of the anæsthetic, as has been successfully done in cases of eclampsia.

Could the *curara* poison, if injected into the veins, or into the sub-cutaneous cellular tissue, in sufficient doses frequently repeated, modify the convulsive influence of the virus of rabies, by acting on the nervous system intermittingly? Curara has, however, been used without any satisfactory result in tetanus, although the failure may perhaps, in great part, be ascribed to the manner it was administered. We have seen how far opium and morphia could, by inducing sleep, quiet the nervous excitement, and prevent the return of the convulsions. We have seen how chloroform-inhalations and curara could arrest the spasmodic convulsions from their special action on the nervous system. But by so doing, we only follow the indications furnished by the principal symptoms of the disease. Let us inquire, however, whether there be not a specific remedy, an antidote of rabies. In this complaint the saliva seems to be the sole vehicle of the virus, as shown by experiments made by veterinary surgeons. Attempts have naturally been made to modify by means of mercury, which acts on the salivary glands specially, the salivary secretion, and the composition of the blood. According to Van Swieten, mercurial preparations have

been of service in hydrophobia. The Chinese believed the following formula to be *infallible*:—

℞ Musk . . . . . half an ounce.
Native cinnabar . . } of each, five drachms.
Artificial cinnabar . }

These substances were rubbed down together to an impalpable powder, and were then given suspended in a spoonful of rice spirit. Calm sleep and copious perspiration came on after two or three hours; otherwise, a second dose of the powder was given, and a cure was considered as sure to follow.

Van Swieten tells us also that mercurial preparations alone, without musk, have proved useful in rabies, and mentions as a proof of his assertion the fact, that of 200 beasts bitten by mad dogs, and to which turbith mineral was given in doses of from twelve to twenty-four and forty-eight grains, not a single one died. He relates also, that a young man who exhibited all the symptoms of confirmed rabies after being bitten by a mad dog, got well by taking every night, for three nights, a drachm of turbith mineral (the yellow sub-sulphate of mercury) and a small quantity of theriaca.[1]

I mention these cases because they seem to me to possess great value, as they are given in the work of Boerhaave's commentator; and they should induce us to try again, in dogs that have been inoculated with rabies, the effects of mercurial preparations. Should some improvement follow their use, we should not hesitate to prescribe during the incubation-stage, and at the outset of the period of invasion, mercury in large doses, in order to arrest in the human subject the progress of rabies.

You may perhaps be surprised, gentlemen, that I should dwell so much on the treatment of rabies, especially at a time like the present, when no faith is put in the measures which used to be vaunted in this complaint. But the fact that it is almost universally regarded as incurable, compelled me to bring to your notice methods of treatment recommended by trustworthy practitioners. And, rather than I should authorise you, by my excessive reserve, to remain perfectly inactive in a case of rabies, I have preferred to bring before you, and thus induce you to repeat, trials made by our predecessors, and even to encourage you to make fresh trials, by pointing out to you the way which seemed to me the best.

In presence of a complaint which terminates constantly in death, the practitioner's duty consists in boldly trying everything.

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[1] [*Theriaca*, according to Bouchardat, is a chaotic combination of stimulants, tonics, astringents, antispasmodics, and principally *opium*, one grain of which is contained in about every drachm of the preparation.—Ed.]

[The concluding words of the above Lecture induce me to append a note on a method of treatment of hydrophobia which occurred to me when thinking on this subject, but which, as I have since learnt, was long ago suggested by Dr. Physick and Mr. Mayo, and approved of by Dr. Marshall Hall. The measure to which I allude is that of *tracheotomy*. It has never, I believe, been tried on a man, but Sir T. Watson, who is opposed to it, states that it was once tried upon a dog by Mr. Mayo, and failed. This unfavourable result in the case of the dog should not, however, lead us to anticipate the same ill-success in the human subject. For I think that Professor Trousseau has, in the above Lecture, clearly established the fact that rabid dogs are differently affected from human subjects; that while, in the latter, the inability to swallow arises from spasm of the respiratory organs, in the former, it is due to paralysis of the muscles about the jaws; and that, whereas a man who is attacked by hydrophobia generally dies of *asphyxia, sudden* or *gradual*, mad dogs, on the contrary, die in every instance of sheer exhaustion. The same virus, in a word, produces effects which are, to a certain extent, different according as the subject is a man or a dog. That, in the human subject, the chief danger to life in hydrophobia arises from difficulty of breathing, is accepted by most authorities. Thus, Romberg (Diseases of the Nervous System, vol. ii. p. 133, Dr. Sieveking's translation) states that 'the individual, on attempting to drink, is seized with a peculiar difficulty of swallowing, which consists *less in an incapacity of swallowing than in an impediment to this function by a difficulty of breathing;* the patients uniformly describe their sensation as one of *suffocation* and strangulation when they swallow, accompanied by great anxiety, which is increased at every repetition of the experiment. *Sobbing inspiration* precedes, the shoulders are elevated, and the epigastrium is tumid, *as in an attack of asthma.*'

The italics are my own; but nothing can be clearer than this passage. The difficulty is one of breathing; the patient cannot drink because he cannot breathe; a spasm of the respiratory organs is the *fons et origo* of the train of morbid phenomena.

Now, if we turn to the pathological appearances met with in the bodies of persons who have died of hydrophobia, we find that they all point to asphyxia. In a note to his translation of Dr. Abercrombie's work, ed. 2, p. 578, Dr. Gendrin of Paris remarks—'I have seen several hydrophobic patients, and I have been present at the examination of the bodies of a great many more. It is only a few months since I observed a case of this horrible disease from its first symptoms to its fatal termination. I have never seen the least trace of inflammation or of lesion whatever in the cerebro-spinal organs or the ganglionic centres. The only lesion which I have found is a considerable development, mostly inflammatory, of the mucous follicles of the base of the tongue, the pharynx, and the upper aperture of the larynx. Hydrophobic patients *die of asphyxia*; dissection shows, in their bodies as in those of persons who have died of tetanus, pretty marked congestion of the pulmonary veins, a general congested state of the principal viscera, and especially of the brain, and liquid blood of a dark red colour in the blood vessels.'

The object of tracheotomy, I take it, is not merely to allow time for the parts to recover themselves, but to prevent the immediate risk of death by asphyxia, and also to allow time for the physician to act, to try and subdue or remove, by the subcutaneous injection of morphia or of atropia, the excessive nervous excitability of the patient. As to the operation itself, it can be easily performed while the patient is under the influence of chloroform.

Another surgical measure which has been recommended by Dr. Brown-Séquard (see his Lectures on the Central Nervous System, Appendix, p. 261) might be tried also, namely, division and excision of a portion of the nerve or nerves distributed to the bitten part. The arguments used by this eminent physiologist and physician in favour of his suggestion, are: '1st.—That an alteration takes place in the part of the body that has been bitten by a rabid dog, before the convulsive and other phenomena of hydrophobia appear. 2nd.—

That the convulsions of hydrophobia occur by fits following a kind of *aura* (pain or other sensations) starting from the wound of the bite or its cicatrix, (which very often then gives way, and is replaced by a bleeding or suppurating wound).'

Dr. Brown-Séquard admits, that there is a poisonous principle in the saliva of rabid individuals, but he thinks that it is in consequence of changes produced locally in the nerves wounded by the bite, that the phenomena of hydrophobia occur. He mentions in support of this view, a most interesting case communicated to him by Dr. Stokes, of Dublin, and which occurred in the practice of Dr. Stokes's father. A tourniquet was applied to the bitten limb of a patient attacked with hydrophobia, and the symptoms quickly improved, and even seemed to cease altogether. The surgeon then proposed to his colleagues to amputate the limb, but they declined to assent to the operation. It was ascertained several times, that so long as the tourniquet was applied to the limb, no convulsions occurred; while they came on as soon as the tourniquet was removed. As from a fear of inducing gangrene, the tourniquet was not constantly applied, the spasms returned, and the patient ultimately died.—Ed.]

END OF FIRST VOLUME.

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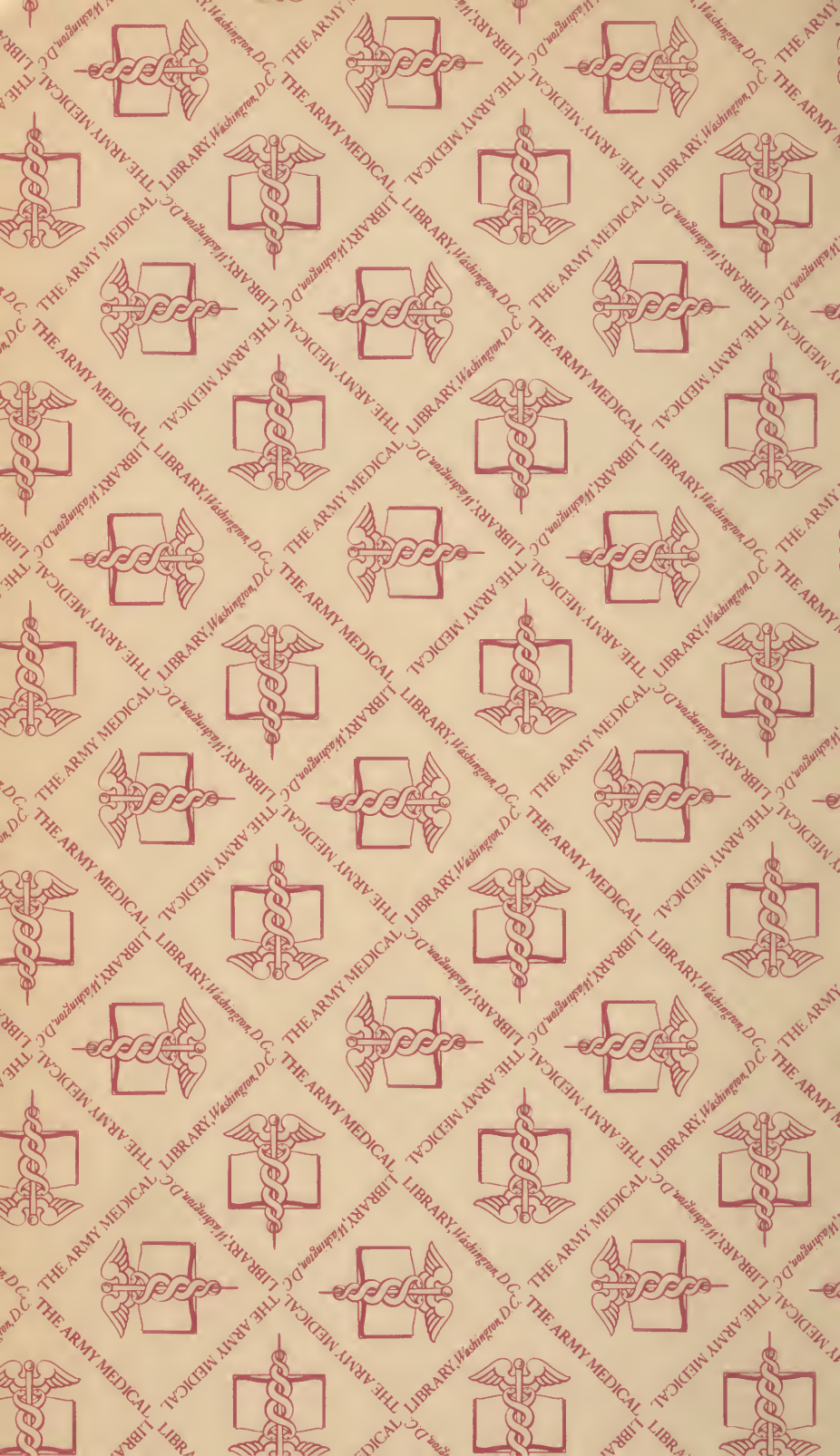
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